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ROLE OF ENVIRONMENTAL FACTORS
IN THE DEVELOPMENT OF
CHRONIC LUNG DISEASE IN
SMOKING AND NON-SMOKING
ADOLESCENTS

FINAL REPORT
NOVEMBER 30, 1986

URBAN AIR ENVIRONMENT GROUP

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NOVEMBER 30, 1986**

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EXECUTIVE SUMMARY

The Urban Air Environment Group, Department of Medicine, McMaster University, has completed two studies of respiratory health in a cohort of over 3200 Hamilton adolescents, the first beginning in 1978. In the report to our sponsoring agencies (Health and Welfare Canada, and the Ontario Ministries of Health, and Environment) we concluded after the first three-year study that smoking by the mother in the home, and hospitalization in infancy with respiratory disease were associated with increased symptoms of chronic lung disease, and measurable changes in lung function indicative of increased airflow obstruction. Air Pollution, as assessed by exposure to suspended particles, showed little or no effects on respiratory symptoms, and only slight effects on lung function.

PURPOSE

The purpose of the second study, the subject of this Report, was to examine the role of environmental factors and their interaction with the onset of smoking, in the development of chronic lung disease in adolescence.

In this study, we followed children from the same cohort, completing measurements on them once in 1983-4, and again in 1985-6. For reasons associated with early termination of funding, only the 1983-4 data have been analysed: these were gathered in what we call "Period IV".

We examined three classes of factors which might predispose adolescents to demonstrate early signs of chronic lung disease:

- i) Respiratory morbidity before the onset of smoking;
- ii) Environmental factors before the onset of smoking;
- iii) Interaction of environmental factors concurrent with smoking.

RESULTS

i) "Wheeze" and "cough" in females, and "asthma" and "cough" in males, determined at the time of the first study, predisposed them to show early signs of chronic lung disease, as reflected by a decrease in lung function measured in Period IV. Even when adjusted for height, the best predictor of lung function in adolescence was the same variable measured several years previously. This supports the view that a child with "low" lung function at age 10 will have "low" lung function at age 13.

ii) Maternal smoking during the time of the first study was associated with a decrease (in Period IV) in the most commonly

used index of lung obstruction (FEV_1/FVC) in males, but not females. There was only slight evidence of an effect of early exposure to air pollution variables (particle size and concentration, and sulphur dioxide). We found that exposure to suspended particles was associated with an increase in one variable indicative of mild airflow obstruction, and only in females. No other environmental factors measured in the first study was associated with any decrease in lung function in the current study. It must be noted, however, that some strong positive associations were found between air pollution and lung function variables, especially with fine particles and sulphur dioxide in males. We propose to investigate this anomalous finding further, if we can find suitable funding.

iii) Rates of self-reported smoking found in this study were higher than those recently reported, and rates in females were approximately twice that reported in males: for example, in 16-year olds, 50% of females were regular smokers, as compared to 25% of males. Self-reported smoking in Period IV was associated with increased lung obstruction, but only in females. Strong interactions were found in Period IV between self-smoking and wheezing in females, and self-smoking and cough in males. Only one air pollution measure from Period IV (suspended particles) was associated with a decrease in Period IV lung function in females, and it was the same one as had been observed in (ii) above. Positive associations between air pollution and respiratory health variables in males were found in the Period IV data similar to those described in (ii) above.

CONCLUSION

Prevalence of smoking in the early teens, particularly in girls, was found to be much higher than previously reported. Early respiratory morbidity, smoking by the mother, and current self-reported smoking can be shown to be associated with early signs of chronic lung disease in adolescents. On the other hand, we have little evidence that air pollution at the levels observed during the time of our study in Hamilton had any influence on the development of chronic lung disease.

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FOREWORD

The following is the final report of a study proposed to Health and Welfare Canada, National Health Research and Development Programme (NHRDP) in July, 1981, and proposed to the Ontario Ministry of Health, Strategic Research and Manpower Planning Division (OMH) in October, 1981. The report also includes the description of work supported by the Ontario Ministry of the Environment, Policy and Planning Branch (OME), carried out as project # 119PL. Approval of the project as described in the proposal was received in December, 1982.

Purpose and Design of Study

The project was proposed as a fundamentally health-related study to examine the role of environmental factors and their interaction with the onset of smoking, in the development of chronic lung disease in early life. From our previous study, we had a comprehensive set of information obtained from a cohort of young people obtained in 1979, 1980, and 1981. At that time we randomly selected over 3000 children (mean age in 1979 of 9 yr) from the Hamilton public school system. Data gathered in each of the three years included respiratory health of the child and his family members, pulmonary function tests of the child, and exposure to ambient suspended particles (TSP, Fine Fraction), and sulphur dioxide. In addition, a broad range of socio-economic, family smoking, and housing information was also obtained with respect to these children each year.

The current study was designed to follow the cohort of children for a period of a further five years, with a nine-month period at the end for final data analysis and reporting. Thus the study was proposed to start in July 1982 and continue until April 1988. During the five-year data-gathering period, it was intended that 2/3 of the children would be examined on three occasions, and one-third would be studied twice. The measurements to be carried out would be a subset of those measured previously for pulmonary function, a subset (with some additions) of those measured for respiratory health, and similar measures of air quality. In addition, the degree of smoking was to be assessed in the young people by self-report, strengthened and validated biochemically by a measure of estimated alveolar carbon monoxide.

Progress

As indicated above, the study began in December 1982, and the measurements in all of the children were completed for the first "round" of this project by December 1984, and by this time two progress reports had been submitted, showing that we were keeping to our proposed schedule. We were obliged to reapply for funding in 1983, and at that time funds were again made available, but for one year only. As a result of a site visit made to us by the two health agencies in May 1984, we were

requested to add to our validation of self-reported smoking a measurement of saliva cotinine, and to spell out in greater detail our specific plans for analysis of the data from the current study. A 20-page response to questions raised at the site visit was delivered to the agencies in June, 1984. A renewal application for the funds to complete the study was prepared, incorporating the suggestions made by the site visit team, and was submitted to both agencies in the Fall of 1984. In May of 1985 we were informed by NHRDP that they would not fund data-gathering beyond June 1985; that we were to cease gathering new data at the end of June, 1985, and that as far as NHRDP was concerned the project was to be terminated, all funding to cease by April 1986.

Appeal of Termination

A letter of appeal was sent to NHRDP, and we were told that our project had been terminated because "it was no longer relevant to the aims of NHRDP". Relevancy ratings of projects submitted to NHRDP, we were told, are performed in-house, and result in an administrative decision not subject to appeal. Furthermore, the evidence used to make such a decision was not available to us. We were told that by increasing the emphasis on smoking validation our study was now "just another smoking study", which was no longer of concern to Health and Welfare Canada.

Support by Province of Ontario

Although the Ontario Ministry of Health was sympathetic to the continuance of our study, they did not have sufficient funds to bear the whole cost of the study. We were particularly distressed, because when notice came to stop the study in May 1985, we were more than 1/3 of the way through the second round of data gathering (still on time to our original schedule). We made an extraordinary appeal to the Ontario Ministry of the Environment for sufficient funds to meet the shortfall created by the pullout of NHRDP, and we received the full funding (data gathering until April 1986) from OMH that had been promised to us on the basis of the 1984 submission. Thus we were able to obtain the full set of data on our cohort (now approximately 1800 young people) for the second round of the current study, and for the fifth time in all. We had sufficient funds to gather the data, but none to analyse the results of the Period V data. In September, 1986 the OME generously provided the funds to carry out the analysis, which should be completed by September 1988.

Effect of Termination on Design

Termination of this study after only one period completely destroyed the design of this study, which depended on the ability to make at least two, and optimally three sets of observations on the cohort, during which time we were carefully

assessing smoking behaviour. Since respiratory symptoms were being assessed by a questionnaire now being administered to the young person, instead of their parents, as had been the case in the past, the questionnaire data could not be compared directly to that previously obtained. This problem had been foreseen, its extent evaluated and documented in the Proposal for this study. Thus it was also essential to the design of this study that at least two sets of measurements be obtained on the cohort using the new questionnaire, so changes in prevalence of symptoms could be established and used as dependent variables. It was for these reasons that we fought so hard to be able, at the very least, to complete the Period V measurements.

Analysis Approach Dictated by Change in Design

Our approach to the analysis of the data being presented in this report has thus been dictated by the limitations of the data we have been allowed to gather and analyse to this point. We have not been able to use the Period IV respiratory health data as dependent variables, except in a very limited way. We have used them as covariables in the analysis of pulmonary function, making use of the data we had previously obtained. The thrust of the analysis has been to analyse the effect, on various indices of pulmonary function, of the independent environmental and self-smoking variables we have measured; adjusting pulmonary function for height separately in males and females, and allowing for the effects of the value of pulmonary function previously measured in the same child.

Features Unique to This Study

It is true that our two studies combined have probably been the largest and most expensive studies of respiratory epidemiology of children ever undertaken in Canada, and rank equally in size to the largest carried out in the world. Size and cost alone, however do not merit respect. Because of the design of the studies, and of the particular mix of skills available in our group, there are several features of our two studies which are unique to the field of respiratory and air pollution epidemiology:

- * Both the health and the environmental data generation have been carried out by the same group.

- * Documented quality control procedures have been used in all data collection modes.

- * The best air pollution exposure estimates yet obtained for an epidemiological study, because of:

- Network size (largest number of monitoring sites/child studied)

- Network design (site locations in residential rather than industrial areas)

- Exposure estimation (a separate exposure value obtained

for each child in the study based on the location of the child)

-Close control of the measurements by our own group

* The most comprehensive battery of pulmonary function tests ever administered in a field study in Canada.

* The most comprehensive set of data on "confounding variables" ever obtained in an air quality study.

* The only biochemically validated data on self-reported smoking behaviour obtained in a large group of Canadian adolescents.

* One of the very few sets in the world, and the only Canadian set of data in a large cohort (approximately 1800) of growth of pulmonary function in children, covering 5 measurements over 7 years.

The combination of these features in one study is also in itself a unique feature of our work, and we are very grateful to the agencies which have supported us, and have allowed us to make this contribution in the field of air pollution epidemiology in Canada.

SUMMARY

In 1979, 1980, and 1981, in Hamilton, Ontario, we measured respiratory health and pulmonary function in a cohort of over 3000 school children whose ages were from 7 to 10 yr in the Fall of 1978. From 1979 to 1986 we also measured air quality: Total Suspended Particulate (TSP); Particle Size Distribution (PSD), expressed as Aerodynamic Mass Median Diameter (MMD), or concentration of the Fine Fraction (FF); and Sulphur Dioxide concentration (SO₂).

We have obtained respiratory health and pulmonary function data on those still available from this cohort (over 2000), and who subsequently were adolescents over the period 1982-1984. Simultaneously, estimates were obtained of their smoking habits. The validity and repeatability of these data have been measured and are found to be within acceptable limits.

The purpose of the present study was to answer three questions:

- 1) Does respiratory morbidity, present in children before the start of smoking, as measured by the prevalence of respiratory symptoms and the presence of pulmonary function abnormalities, predispose a child to develop small airways disease after smoking has started?
- 2) Are there adverse environmental factors present during childhood which lead to the development of small airways disease on the start of smoking?
- 3) Are there concurrent environmental factors which appear to interact with cigarette smoking in the development of small airways disease?

In response to the first question we offer the following:

All pulmonary function values (except for FEV₁/FVC, and MET, which are inherently independent of lung size) were adjusted for the height of the subject, based on regression equations developed from our own data. The strongest predictor of any measure of pulmonary function obtained in Period IV (the current study; 1982-1984) was the value obtained in the same subject in Period II (1980). This supports the view that a child with a "low" pulmonary function at age 10 will still likely have a "low" pulmonary function at age 13.

The presence of wheezing in Period II and III in females, was the variable most consistently associated with a decrement in pulmonary function in Period IV. In males, asthma in Period III was most consistently associated with a decrement in pulmonary function in Period IV.

In those pulmonary function variables thought most likely to represent "small airways function" (MEF₇₅, and FEF₇₅₋₈₅)

there was evidence of a decrement, in association with "cough", and "wheeze", in females only. None of the other health variables previously referred to were found to be associated with any decrement in males or females.

Self-smoking in Period IV was included in the analyses with all of the Period II and III variables described above, and was not found to be associated with any decrement in height-adjusted pulmonary function in males or females.

Thus it appears that: previous level of pulmonary function, previous wheeze, to a lesser extent cough and asthma in males, and, to some extent, previous maternal smoking; are the factors in early life associated with the development of small airways disease in adolescents.

In answer to the second question we offer the following:

None of the air quality variables measured (TSP, FF or SO₂) in either Period II or Period III (1981) were associated with a decrement in pulmonary function in males in Period IV, and in females in only one measure, MET, in Period III only, was there an effect which could be considered a decrement in function, and it was associated only with TSP.

Maternal smoking in Periods II and III was associated with a decrement in the most commonly used index of pulmonary obstruction (FEV₁/FVC) in males, but not females in Period IV.

None of the environmental variables measured were associated with a decrement in MEF₇₅ or FEF₇₅₋₈₅ in Period IV, and thus we did not see evidence of decrement in "small airways function" in males or females.

The response to the third question is as follows:

The environmental variables we studied in Period IV included TSP, FF, and SO₂, as well as maternal smoking. At this time the Period IV SO₂ data are not processed sufficiently for analysis, and our comments will be confined to the other variables. Only one of these other variables were associated with any decrement in pulmonary function in males or females in Period IV, and that was TSP on MET in females only.

Respiratory health variables (cough and/or wheeze) observed in Period IV were associated with a decrement in several pulmonary function variables measured in Period IV in both males and females. Asthma was associated with a decrement in males only.

Self-reported smoking in Period IV was associated with a reduction in FEV₁/FVC, but only in females. There was a

strong interaction between self-smoking and wheezing in its effect on pulmonary function in females, and also between self-smoking and cough on pulmonary function in males.

Indicators of "small airways function" showed no decrement in association with environmental factors in Period IV.

We interpret these findings to indicate that at ambient levels of TSP, fine fraction, or SO₂ measured over the course of this study, we have no evidence of "small airways disease" associated with these indicators of air pollution. In females only, evidence of "small airways disease" was associated with wheeze in earlier years (Periods II and III), and with current (Period IV) cough. These latter associations were not found in males. Interactive effects in Period IV were found in males; between smoking and cough, and small airways function; and in females between smoking and wheeze, and small airways function.

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1. INTRODUCTION AND BACKGROUND

1.1 Nature of Research Question:

In 1982, we began a study of the pulmonary function of children in the age interval 10-13 years. The subjects studied were a cohort that had been assembled in 1978 to examine the effects of the environment on children's respiratory health. The purpose of our new study was to gain new insights into the early development of lung disease, especially chronic air flow obstruction. Air flow obstruction has been shown by Fletcher to have a gradual onset during adult life with a proportion of those who smoked showing a much greater rate of decline of FEV1 (1). Burrows (2) in a cross-sectional survey, showed the same pattern of a gradual decrease in FEV1 during adult life with some smokers demonstrating a much greater rate of decline. Burrows also showed that those who had some history of child respiratory illness, especially of the asthmatic type, had lower pulmonary function in adult life. These two studies raised questions about what were the predisposing factors that led some smokers to develop this greater than average rate of decline in pulmonary function and also how soon after smoking one could recognise the smoker who was at risk of potential pulmonary disability.

1.2 Studies of the Consequences of Respiratory Childhood Disease on Lung Function

The only prospective study from infancy into adult life that has looked at the effects of smoking unfortunately did not utilize any pulmonary function measures. They did show that respiratory illness before the age of two increased the prevalence of a cough at the age of 25 in addition to the effect of personal smoking (3). Focusing on infancy, Leeder showed that respiratory illness before the age of 5 had an effect on peak flow measured at the age of 5 and that the earlier and the more frequent the attacks, the greater was this decrement (4). He also showed that a history of respiratory infection in the first year of life increased the prevalence of wheezing at age 5 (5). Bland (6) described the relationship between respiratory symptoms at the ages of 11 and 14 and the occurrence of asthma or respiratory infections before the age of 5. These illnesses prior to the age of 5 had the most marked effect on the prevalence of wheezing in later years. The importance of wheezing as a marker of air flow obstruction has been pointed out by Weiss (7). We have also found that children in the age interval 10 - 15 years who have a history of current wheezing, even after excluding those with a history of asthma, will have an increased prevalence of pulmonary function abnormality compared to those without the symptom (22% with wheezing have an FEV1/FVC ratio less than 75% compared to 13% without wheezing). Therefore, although the study by Bland did not report on pulmonary function, the presence of wheezing can be used as an

indicator of underlying pulmonary function abnormality. Woolcock, using retrospective histories of early respiratory illness, has also shown a relationship between the occurrence of this early history and the presence of pulmonary function abnormality, as measured by a decrement in the V50 with 9 and 13 year old students in Australia (8). In our review of the literature, Woolcock has been the only author to integrate this information about early childhood respiratory illness with the effect of self-reported smoking in older students.

1.3 Studies on the Early Effects of Cigarette Smoking

A number of studies have now been published describing the relationship between early cigarette smoking and changes in pulmonary function. Seely (9) described a reduction in both V50 and V75 in male high school students who smoked more than 15 per day and female high school students who smoked more than 10 per day. Smoking was not associated with any change in FEV1 in the study by Woolcock (8), and there were small and somewhat inconsistent relationships shown between smoking and pulmonary function. Male smokers who had a previous history of bronchitis before the age of 2 showed a reduction in V50 if they smoked. In contrast only female smokers who had no previous respiratory history showed significant reduction in V50. However, the lack of any significant effect in the female smokers with previous respiratory illness may well be due to the small numbers in that particular category which reduced the power of the analysis below that necessary to show a significant difference if in fact it existed. Two studies by Tager (10,11) have reported on the early effects of personal cigarette smoking. The first study described a relationship between maternal and self-smoking and over a five year period, found that the children who smoked had a much lower expected rate of growth in their FEV1 than those who did not smoke (1436 ml for non-smokers against 1094 ml for smokers). If in addition, the mother smoked, the expected rate of growth decreased to 993 ml over a five year period. In the later study by Tager, similar findings were found in terms of a significant decrease in the rate of growth of FEV1 and FEF2575. The changes which they observed were seen mainly in those subjects starting smoking at the age of 15 or more. A further study from the United Kingdom by Adams (12) was longitudinal in design, over four years, of students, mean age 13 years at the start of the study. No significant difference could be found in pulmonary function after one year of smoking but after two years, those who smoked more than seven cigarettes a day had a significantly reduced V50 compared to non-smokers. After three years, the peak flow of smokers was significantly reduced compared to non-smokers. These authors also showed an increase in the slope of Phase III of the single breath nitrogen washout test in smokers after two years of smoking.

Although the studies by Tager and Adams were not available to us at the initiation of our own study, they still leave unanswered some of the questions that we posed in our original grant

submission. They did show that using measurements derived from the flow-volume curve, one could see changes with early smoking, although their subjects were older than ours and were therefore likely to have a heavier and more prolonged experience of smoking.

1.4 Research Questions of Present Study

In our grant submission, based on the review of the literature, the following research questions were posed for the current study:

- 1) Does respiratory morbidity, present in children before the start of smoking, as measured by the prevalence of respiratory symptoms and the presence of pulmonary function abnormalities, predispose a child to develop small airways disease after smoking has started?
- 2) Are there adverse environmental factors present during childhood which lead to the development of small airways disease on the start of smoking?
- 3) Are there concurrent environmental factors which appear to interact with cigarette smoking in the development of small airways disease?

2. DESIGN

2.1 Epidemiology

Background

The sample that we studied was one originally drawn in 1978 for a study of the effects of environmental factors on the respiratory health and the pulmonary function of school children. The sampling frame was all public elementary schools in Hamilton Ontario. The city was divided into four quadrants as shown in Figure 2.1: West Upper (WU), East Upper (EU), West Lower (WL), and East Lower (EL). Within each quadrant schools were randomly selected until in each quadrant, 800 children in grades 2, 3, and 4 had been included. In the second year of the study, all new children at these schools that were in the same age interval as the initial cohort were also included in the study. Three new schools from the area of the city with the highest industrial pollution (the Industrial Core; "IC") were also entered in the second year of the study and all children within the same age intervals as the initial cohort were added.

Consent for the study was obtained from the parent of each child. The consent rate in each year of the study exceeded 95%. Each child was studied by means of a questionnaire administered

to a parent by an interviewer, and by pulmonary function testing in the school; yearly for three years. We refer to the three measurement years of the 1978 project as Period I (1979), Period II (1980), and Period III (1981). Epidemiological data gathering for this study concluded in January 1982, the study itself officially terminating in June 1982.

We then continued to study this cohort in order to answer the research questions detailed in the previous section. Consent was once again obtained from the parents for their child for the remainder of this second study which was initially envisioned for a five year period with a maximum of three observations on each child. Of the 2692 children still available for the study in June 1982, parental consent was obtained for 2,137 (79.4%).

Original Design

The original design for this study intended it to begin in July 1982, and that data collection would continue for 5 years. During the data collection period all students in the study were to have been tested on three occasions spread more or less evenly over the available time. A nine month period at the end for analysis and report preparation would then have taken the study to completion in April 1988. However, notification of approval of this proposal was not received until December 1982, and thus the beginning of testing of the children did not begin until December 1982. This first complete round of testing in the present project was completed on schedule in December 1984, and was in fact the fourth time we had tested these students: we refer to this round of testing as "Period IV".

Alteration of Original Design by Health and Welfare Canada

Testing recommenced in January 1985, but by May 1985 we were instructed by Health and Welfare Canada to terminate data gathering at the end of June 1985. However, through funding made available by the Ontario Ministries of Health, and Environment, we were able to continue our measurements until mid-June 1986, and were able to finish the second round of testing in this project, which we refer to as "Period V". Approximately 1800 students in all have 5 sets of test data covering the years from 1979 to 1986.

Health and Welfare Canada stipulated that a final report be written on the basis of data collected until June 1985. Since this represented a departure from the original design approved by Health and Welfare Canada in December 1982, it will not be possible to meet the objectives of the original design as planned. Only about 1/3 of the data for Period V was collected by June 1985 (well within our original schedule), and so the analysis must be restricted to measurements made in Period IV only. No longitudinal analysis beyond relating the earlier study data to Period IV can be undertaken.

All the testing of the study was performed in the schools. Prevalence of respiratory symptoms was estimated by a self-administered questionnaire. Validation of reported smoking behaviour was provided by measurement of expired carbon monoxide. The technique that was followed was modified from that by Vogt (13).

Pulmonary function testing was performed in the school. The apparatus used both in the present and in the previous study was an Hewlett-Packard pneumotachograph based Pulmonary Calculator System. In this system, flow is measured by the pneumotachograph and volume is derived by integration of the flow over time by a small computer. Computations were performed internally and a graphic output of records was obtained for each child at the time of testing.

At the time of testing, the tester also recorded and coded the presence of upper or lower respiratory infection so as to allow any analysis to take the presence of these infections into account.

The reproducibility of both the questionnaire information and the pulmonary function testing was estimated by selecting randomly five per cent of the subjects within each school and retesting these subjects at the conclusion of the period of study in that school. All tests, including the respiratory symptoms and smoking questionnaires, the carbon monoxide test and the pulmonary function test were repeated on that occasion.

2.2 Air Quality

The objective of the air quality monitoring networks was to provide an estimate of the exposure to ambient suspended particles and sulphur dioxide for each child in the study. We wished to take advantage of the known gradients in distribution of air pollution to be able to use this as an independent variable in our analyses.

Estimation of individual exposure of a child was accomplished by the fitting of quadratic response surfaces to monthly data, and accumulating the monthly predictions for each child's school for the desired exposure period. Coefficients of determination for the bivariate quadratic response model typically have a value greater than 50%, indicating an acceptable fit in most cases.

Extensive air quality measurements have been made in the City of Hamilton since 1978, both as part of our previous study, and as part of the present study. These include measurements of total suspended particulate material (TSP), particle size distribution (PSD), and sulphur dioxide (SO₂) concentration. At the termination of the present project, we operated 14 TSP sites (the Ontario Ministry of the Environment operated 13), nine PSD sites, and 6 SO₂ instruments located at 12 sites over different

periods. The sites that we operated were located to provide air quality data for areas close to our study schools and the residential areas associated with them, in contrast to the location of the OME sites close to industrial areas.

3. METHODS

3.1 Epidemiology

When the tester entered a new school, she would meet with all the students and explain the purpose of the carbon monoxide test. During testing in each school, temperature, barometric pressure and humidity were recorded twice daily to be used in the computation of the pulmonary function variables. In addition, both the carbon monoxide analyzer and the pneumotachograph were calibrated twice daily. For each student, the respiratory symptom questionnaire was administered first. Prevalence of cough was estimated by positive responses to the questions:

- 1) "Do you usually cough in the morning"?
- 2) "Do you cough like this most mornings for as much as three months in a row each year"?
- 3) "Do you usually cough during the day or night"?
- 4) "Do you cough like this most days or nights for three months in a row each year?"

Prevalence of wheezing and asthma was estimated by positive responses to the questions:

- 1) "Does your chest ever sound wheezy or whistling?"
- 2) "Does it usually sound like this most days or nights?"
- 3) "Has the doctor ever said you have asthma?"
- 4) "Have you ever had to use your asthma medication in the last year?"

Prevalence of lower respiratory infection was estimated by positive responses to the questions:

- 1) "When you have a cold do you usually cough"?
- 2) "Is it a chest cough as opposed to a throat cough"?
- 3) "Is it a cough that produces phlegm"?
- 4) "Do you have a cold now, and if not how many weeks ago did you have a cold?"

5) "When you had this cold, did you also have a chest cough?"

Smoking behaviour was estimated using a confidential questionnaire, by positive responses to the following questions:

- 1) "Have you ever smoked"?
- 2) If so "Have you smoked at least one cigarette a week for a period of six months"?
- 3) If so "How many cigarettes do you usually smoke in a week"?

The student was also asked at what age the first cigarette was smoked and if a regular brand was smoked. The questionnaire also requested information on maternal smoking and the number of other smokers in the household. The student was then weighed and measured using a standard stadiometer with the student in stocking feet.

The tester would then re-emphasize to the student the purpose of the carbon monoxide test. In this method, the student inhaled, held his breath for twenty seconds and then exhaled roughly a quarter to a third of the vital capacity and collected the remainder in a plastic bag from which, after allowing time for equilibration, the carbon monoxide was measured by a carbon monoxide analyzer (Gas-Tech, HEC-231). Ambient carbon monoxide concentrations were also recorded at the same time. To maximize the validity of this reporting we employed the "pipeline" technique, as described by Evans (14). The technique, as originally described, was a "Bogus Pipeline" in which only part of this sample was informed of the ability of the particular biochemical test to validate reported behaviour. Because Evans, (14) showed that giving this information increased the reporting rate, we decided to inform all participants in the study of the nature of the test, and specifically, that it would identify recent smoking.

Pulmonary function was then measured, with the student standing and non-supported. The subject performed a forced expired maneuver from total lung capacity to residual volume followed by an equally rapid inspiration to total lung capacity. Vital capacity maneuvers were recorded and, to be acceptable, at least two forced vital capacities had to be within 5% of each other and the FEV1 derived from those tests had to be within 10% of each other. Computations were performed internally and a graphic output of the record was obtained for each child at the time of testing. For the purpose of measurement, the calculator selected the test with the largest sum of FEV1 plus FVC. Both inspiratory and expiratory flow-volume loops were obtained. From these flow-volume loops, the following variables were also measured; peak flow, MEF50, MEF75, MEF25-75, MEF75-85, MET.

Statistical analyses were performed using sub programs in the Statistical Package for the Social Sciences (22). These included descriptive statistics, comparison of sample means and comparison of observed with expected frequencies. The effect of one or several independent variables on a continuously distributed dependent variable was also performed by multiple regression using a sub program from the same package. The actual procedure for each particular multiple regression analysis will be described in the section on data analysis. Calculation of confidence intervals for estimates of prevalence was performed by calculating the binomial confidence intervals, given the number of successes and the number of trials, by first relating the binomial distribution to the incomplete beta function and then solving two incomplete beta function equations. IMSL algorithms (15) were then employed to solve these equations and derive the confidence intervals. Adjustment of level of significance for multiple comparisons was performed by the method of Tukey (16), in which the conventional level of significance (0.05) is divided by the number of questions being investigated. Correction of measures for agreement due to chance was performed by Cohen's Kappa (17).

3.2 Air Quality.

3.2.1 TOTAL SUSPENDED PARTICULATE

Reduction of Network Size.

We have measured TSP levels across the city of Hamilton continuously since 1978, when we began our "School Children's Study" project. In July 1981, when we made the first application for funding of this project, our TSP sampling network was made up of 17 high-volume (hi-vol) samplers. We attempted to reduce the size of the network by step-wise analysis of predicted exposure values derived from our response surface modelling procedure. Using seasonal data, we determined which sampling site would have the least influence on the network results, removed its data from the analysis, and proceeded again to identify the next location until 8 samplers were removed. Unfortunately, this process showed that the most "removable" sites differed from season to season. Indeed, even the most consistent sites appeared on only three out of the four seasonal lists, and then with different rankings. This work concluded with the removal of three of these sites. However, to operate with a further-reduced number of samplers would at any one time have required activating and de-activating sites on a 3-monthly basis, which ultimately would have been more labour-intensive than maintaining all sites active during the year. Since our exposure calculations are based on monthly averages, it was necessary to maintain the network in a configuration of 14 sites. This proved to add little to the burden of operating the network except for a small increase in weighing and data entry time.

3.2.2 PARTICLE SIZE DISTRIBUTION

Our 9-site PSD sampling network has a history similar to that of our TSP network. However, we have maintained its size as its coverage of the area is thinner. Additional details of the operation of our TSP and PSD sampling network are given in (18), "A Field Guide to Air Quality Assessment, by W.Furlong".

With the resources provided to us by the Ontario Ministry of the Environment (Project 119 PL) we were able to reanalyse the PSD data from 1979, 1980, and 1981 and express them in the form of "Fine Fraction (FF)" and "Coarse Fraction (CF)", the division point between the two being at 3.3 μ m aerodynamic mass median diameter (AMMD). Having expressed the data in this fashion it was then possible to compute response surfaces and to carry out exposure estimation for all of the children in the first study based on FF and CF. Exposures were then attached to the childrens' files, and used as independent variables in the assessment of health effects using multiple linear and logistic regression techniques. Although not in the original proposal for the current study, we undertook the same set of additional computation and analysis for the PSD data in the current study, expressing them as FF and CF and using them as independent variables in the analysis of Period IV respiratory health data.

3.2.3 SULPHUR DIOXIDE MONITORING

Background

In 1977, we carried out a pilot study in preparation for our "School Children's Study" project. During this study, we used the Beckman 906A sulphur dioxide monitor to collect a portion of our air quality data. At that time, the instrument was in constant use by the Ontario Ministry of the Environment, and was still actively sold and supported by Beckman, though new SO₂ monitoring technology was being introduced.

Following the pilot study, we designed the "School Children's Study" project to make use of the Beckman 906A, which was to be calibrated against, and eventually replaced by, the Laser Absorption Spectrometer (LAS) being developed by McMaster's Department of Engineering Physics, under Dr. J. Shewchun (later replaced by Dr. B. Garside). This Engineering Physics laser group was responsible for collection of the SO₂ data, as well as for the development of the LAS. When approval was received for the "School Children's Study" project (in November of 1978), those responsible for SO₂ data collection were to begin monitoring with those 906A's available, followed by the LAS's to complete the network and to keep the 906A's closely calibrated. However, Beckman suspended production of the 906A during 1979, and discontinued its support in Canada during June of 1983. Unfortunately, the laser group was not

able to develop the LAS to the point where it could be operated unattended; thus, at the end of the study we were left with no LAS's. However, with the assistance of the Ontario Ministry of the Environment, we had sufficient Beckman 906A's to fill our network requirements for the present study.

Sulphur dioxide monitoring was terminated by the Engineering Physics group during December 1981, as the data gathering phase of that project was ending, and the one technician who had been maintaining the SO₂ network was leaving. From December 1981, until approval of funding for our present study was received from Health and Welfare Canada (in December 1982), all of the Beckman 906A analyzers used by the laser group were in storage, with the recorders being used for other purposes in the interim.

Data from School Childrens' Project.

We were provided with data on magnetic tape which our colleagues in the laser group told us contained all the results of their SO₂ monitoring work. Preliminary examination of these data showed many inconsistencies which could not be resolved. We knew that the 'raw' data had been processed to remove errors which had to do with variation of chart speed of the recorders, but we did not have access to a complete set of the data coded from the charts, either on tape or in punched card form. Neither were we able to obtain a copy in any form of the listing of the program which was used to 'process' the data to the form in which we received it. Using the description of the method of adjusting the data which they provided us, on a small set of raw data which we had, we could not reproduce the result that had been given us for the same period from their tape.

In the absence of verification of the quality of the data, or a clear 'audit trail' to follow from start to finish, we decided it would be necessary to begin with the original chart recordings, and if possible, reduce them and process them ourselves. Funding for this aspect of our work was sought from, and provided by the Ontario Ministry of the Environment as resources for Project 119 PL.

We located all of the recorder charts covering this period, and all but 20 of the 437 rolls had sufficient identification on them to permit reading and coding. A substantial effort was expended in cataloguing the charts to determine if they were worth coding, and to develop a comprehensive set of rules, criteria and guidelines for data reduction. This is described in detail more fully below.

Current Project

Since September of 1982, we put back into operation as many of the Beckman 906A SO₂ Analyzers inherited from the Engineering Physics laser group as possible. We also obtained a number of other 906A's on extended loan through the kind assistance of the

Ontario Ministry of the Environment. Many of these would have required considerable repair before they could be placed in the field; accordingly, we used them as sources of spares for the other instruments that it was possible to return to running order.

The single technician working on the sulphur dioxide monitoring network was fully occupied with servicing the instruments put into the field, and with attempting to repair others. This proved to be a never-ending task, as we reached the end of the effective life of many of the electronic components in the Beckman 906A. Some concept of the magnitude of the task of reconditioning Beckman 906A's may be obtained from the following list of repairs or procedures which had to be carried out for each instrument (at the minimum):

1. Cleaning each detector cell with acid;
2. Replacement of detector cell electrodes;
3. Replacement of all internal sample line tubing;
4. Cleaning of inlet selector switches;
5. Cleaning of each flow meter;
6. Replacement of electronic components in the range switch and on the amplifier and "water addition" printed circuit boards, in order to meet instrument specifications;
7. Replacement of each selective scrubber. Replacement scrubbers used had been stored by Beckman for a considerable time, causing a reduction in instrument response time even with these "new" scrubbers; and
8. The installation of in-line filters to remove particulate matter from the sample gas stream.

Field Maintenance..

There were a number of problems associated with the electronic circuits, which necessitated their repair or replacement. This occurred in both the amplifier and the "water addition" boards, as well as in the electrical wiring harness itself. Deterioration of instrument thermal insulation resulted in contamination of the electrolyte as well as in loss of temperature stability. Several instruments had to be re-insulated.

Several modifications to the analyzer were also made. It was found that frequent opening and closing of the instrument during servicing was contributing to failure. The temperature control was modified to allow for external adjustment, and a larger water bottle installed to allow a longer interval between refillings. Both of these modifications reduced the frequency of disturbance to the instruments. Both batches of new detector cell electrolyte and reference electrodes obtained from Beckman were found to be defective, so we made up our own electrolyte solution and reconditioned our own electrodes with activated charcoal.

Laboratory tests on improving the performance of the analyzers also investigated a new high bypass flow sampling system to reduce degradation of the sample during low flow in a long line, as well as a prototype automatic capillary flushing system. In addition, a laboratory study of the effect of changing sample flow on indicated levels was carried out, in order to quantify the effect for correction of recorded data. Old recorders have been replaced with new recorders, and chart take-up reels installed on some recorders.

A detailed field manual including site selection, set-up, operation, calibration, maintenance and repair instructions was prepared, which also included an inventory of instruments and recorders (20). An operating log was maintained for all instruments, with a history of performance and repairs.

Coding.

As an important link between the recorded analyzer output and the data on computer file, we have developed a comprehensive manual of instructions and validity criteria for cataloguing, reading, and coding the charts. This has been carried out in parallel with our efforts to recover and restore SO₂ data obtained in 1979-1981 by our colleagues in the School Children's Study. Our SO₂ data reduction technician used the charts from the current project as a basis from which to establish the coding rules and criteria; these have been documented and are presented in Reference 19. The purpose for this careful documentation is to ensure reproducibility of data reduction, so that it is as independent of the level of skill and judgement of the reducer and coder as possible. Quality control is also provided for by random recoding of some of the charts by a second person.

Data Management.

Special hardware and systems were designed to carry out the process of chart reading, coding, data transfer, data editing, and data processing. Several chart reading jigs were designed and built to reduce the effort required to handle charts in the process of raw data reduction. A digitizing system based on a Radio Shack digitizer and TRS-80 Color Computer, was designed and built, and the software written to provide the data in the form decided upon once the coding system had been determined. The coded data were stored on magnetic disk, and were available in printed form, as well as being displayed immediately after coding in graphic form on the coding terminal. Coded data were subsequently uploaded to the McMaster HP 3000 computer via a terminal program and modem implemented on the Color Computer. Not all data were handled in this way, because much of the work was carried out in parallel, and for the sake of expediency many of the charts were reduced 'manually' before the digitizing system became available.

Computer programs to process the coded data were written in

FORTTRAN and executed on the HP 3000. This work was carried out in the main by the same person who designed the coding system, and a complete audit trail was maintained and documented. The output of the processing was a file containing hourly values of SO₂ by site, for all the sites operated. The same system was subsequently used to handle the charts and coded data from the the current study.

Data Display.

We also developed a simplified system of displaying the response surfaces developed from our TSP, PSD and SO₂ data, which allows us to draw "contour maps" of pollution levels across the city. This system can be implemented on a personal computer and printer. Examples of response surfaces for TSP generated in this way are given in Figures 4.2.1 and 4.2.2.

Visualization of the response surfaces allowed us another level of quality control that was implemented before the data were used to calculate exposures for the purpose of relating air quality to health effects. We now know from experience what the distribution of air pollution is likely to be in a given month, and this has allowed us to detect and rectify any major errors which could creep in at one of the many stages required in the processing of data.

4. DESCRIPTIVE RESULTS

4.1 Epidemiology

The characteristics of the cohort studied are shown in Table 4.1.1. There is an almost equal age distribution between male and female subjects. The mean age at the time of testing of the cohort was 12.9 years varying from 12 to 16 years. The age interval of the initial cohort was 7 to 10 years but the age interval increased due to the testing during this current study taking place over a two year span.

Because there had been an attrition of the original cohort since 1979, we compared the prevalence of certain characteristics in 1979 of those that had been lost to follow-up with those remaining in the study. The purpose of this analysis was to determine if there had been a disproportionate loss of certain subjects over this period of time. This comparison is shown in Table 4.1.2. Using the method of Tukey for multiple comparisons (16), the significance level for the nine comparisons that were made is 0.006. Using this level, there were significant differences between the two groups for the prevalence of maternal coughing and smoking and for the occurrence of serious chest infection before the age of two. In addition, there were significantly more lower income families (annual income less than \$10,000 per year) in the group lost to follow-up. However, there were no significant differences in the prevalence of

respiratory symptoms in the child nor of paternal cough and smoking. In Table 4.1.3, it is seen that there were no significant differences in the baseline pulmonary function characteristics.

The prevalence of smoking in each age category is shown in Table 4.1.4. The prevalence of heavy smoking (> than 10 per week) rises from 0% at the age of 11 to 22.7% at the age of 16. Similarly for regular smoking (>1 per week) the prevalence rises from 1.6% at age 11 to 36.4% at the age of 16. These data are further broken down by gender in Figure 4.1.1, which shows the differences in prevalence of regular smoking between males and females in each age group. In each age group, there are more females than males who smoke, the difference becoming 23% at the age 16. 95% confidence limits on these estimates of prevalence are also shown in Figure 4.1.1. In the age groups between 15 and 16 years, the confidence limits are wide due to the relatively small numbers. However, even taking the lower bound of these intervals into account, a steady rise in the prevalence of smoking with age can still be seen. Table 4.1.5 shows the mean difference in carbon monoxide between expired and ambient air in the four smoking categories. The difference in the mean CO difference is least between the never and experimental smoking groups although this difference is significant ($t=3.54$, $p<0.001$). However, the mean CO difference is greater in the group smoking between 1 to 10 per week and greatest in the group smoking more than 10 per week. These differences are significantly greater ($p<0.001$) than the mean CO difference in the never smoking group. In addition, the CO difference in the group smoking more than 10 per week is significantly greater ($t=6.46$, $p<0.0001$) than the group smoking between 1 and 10 per week. The difference between the groups is shown in Figure 4.1.2, which shows the cumulative distribution of carbon monoxide difference for the smoking categories. The distributions for the never smokers and experimental smokers were virtually identical but the differences between these two groups and the other two smoking categories can clearly be seen on this graph. This figure also allows comparison of the four groups for any given cut point of CO difference. For example, if one takes the level of 4 parts per million carbon monoxide, only 6 per cent of the never smokers had a level above this in contrast to 75% of the group reporting more than 10 per week.

Table 4.1.6 shows the relationship between maternal smoking and personal smoking. The presence of maternal smoking is associated with the significantly ($p<0.05$) higher prevalence of regular smoking in both the smoking categories. Combining both these categories, 16.1% of those adolescents whose mother smoked were regular smokers, in contrast to only 7.0% where the mother did not smoke.

Table 4.1.7 shows the reproducibility of the questionnaire data. Using the criteria of Landis (16), any measure of chance corrected agreement (Kappa) between 0.61 and 0.80 is

"substantial" and greater than 0.80 is almost perfect. Using these criteria, therefore the agreement between the initial and repeat responses to the questionnaire is acceptable. Table 4.1.8 shows the details of the reproducibility of the pulmonary function variables. In no case are the repeat estimates significantly ($p < 0.05$) different from the initial estimate.

Table 4.1.9 gives the prevalence of respiratory symptoms in the cohort as a whole, by gender. Note that in females, there was a higher prevalence of cough than in males, but that more males than females were said to have ever had asthma. In all other respects the symptom data obtained were the same for both males and females.

Table 4.1.10 describes the pulmonary function status of the cohort based on our measurements. There was no significant difference between males and females in age or weight, but in all other aspects of measurements differences were statistically significant. Pulmonary function variables most strongly influenced by lung size (such as FVC, FEV1, and PF) were greater in males, but those dependent on flows at lower lung volumes (such as FEF25-75, MEF50, and MEF75) were greater in females. Further details of the cohort may be found in (23), included in the Appendix.

4.2 Air Quality.

4.2.1 TSP MEASUREMENTS

Data from our TSP network are presented in Tables 4.2.1 to 4.2.4 in the form of monthly geometric means. To complement the data provided in the Final Report of Project 6606-1752-53 (dated June 30, 1983), we include all data for the calendar years 1982 to 1985 inclusive. We also include for reference in these tables the corresponding OME data where available. Annual geometric means are also given in Table 4.2.5.

Response surface "maps" are also presented in this Report for annual geometric means of TSP, as Figure 4.2.1. Because these maps were not included in the Report of the earlier project, we include maps covering the period from 1979 to 1985 inclusive. We also include maps of the distribution of particles for the months of May and December for the three year period 1979 to 1981 (Figure 4.2.2), to demonstrate the differences that can be observed from month to month. From these maps, it is apparent that the distribution of particulate matter is similar in appearance for the same month from year to year, but differs from month to month in the same year. This is most likely a result of different patterns of precipitation and prevailing winds, in the different seasons.

Data gathering for the current project was not supported by NHRDP beyond June 1985, but we were fortunate in having the

support of OME in providing funds to complete the gathering of data until June 1986.

4.2.2 PSD MEASUREMENTS

Data for the PSD network are presented in Tables 4.2.6 to 4.2.9 as monthly average Mass Median Diameters. We have, in addition, done the necessary calculations to express the results of the PSD network in terms of a dichotomous sample of Fine Fraction (FF) and Course Fraction (CF), the cut point being 3.3 microns aerodynamic MMD. Monthly geometric means of Fine Fraction ($<3.3\mu\text{m}$) and Coarse Fraction ($>3.3\mu\text{m}$) are given in Tables 4.2.10 to 4.2.13. Data are supplied from 1982 to 1985 inclusive, and were subject to the same funding conditions quoted above.

4.2.3 SULPHUR DIOXIDE

Data from School Childrens' Project.

All of the work has now been completed for the earlier project SO₂ data, which we were forced to reconstruct from the original raw chart records (close to 450 in number) which had been only sketchily annotated. Tables 4.2.14 to 4.2.16 provide monthly arithmetic mean data for the years 1979 to 1981. For technical reasons (relating to the 6-weekly rotations of instruments used in the earlier study) the response surfaces for these data have been generated on the basis of weekly average data, rather than monthly, and exposures have been aggregated from weekly, rather than monthly averages. A complete set of the 151 weekly surfaces is available for inspection if requested from the authors, but has not been included in this Report. The SO₂ exposures from Period II have been used in this Report to examine their influence on Period IV pulmonary function in conjunction with other environmental variables. Rerunning the analyses of the previous study on the basis of exposures to SO₂ is currently in progress.

Current Project.

The two first SO₂ analyzers became operational in two upper city locations in March and April 1983. In addition, the MOE has operated two SO₂ analyzers in the lower city from 1970 to the present. The maximum number of analysers we were able to keep operational in the field at any one time was 6, so that including OME data, we have a maximum of 8 locations from which we can obtain response surfaces. The first new Teco instrument began providing data in the field from November 1984, and a maximum of 4 were ultimately deployed. Table 4.2.17 and Figure 4.2.3 provide information on the dates of operation of sites, and the amount of valid data obtained.

The graphic records of SO₂ data generated by our analyzers have all been annotated for time and location, calibration, and

zero check. A data management and quality control scheme has been designed, documented (19), and implemented, and the data have been reduced from the charts and entered into the computer.

What remains to be done are the computer runs to adjust the data for calibration changes, zero shift, and chart timing errors; to reduce hourly data to daily and then weekly averages; and to compute the response surfaces and then the exposures for the young people in the study.

5. ANALYSIS

5.1 Single associations of current smoking with current respiratory health.

Table 5.1.1 gives the prevalence of respiratory symptoms tabulated by smoking behaviour. For both males and females, self-reported smoking had a significant association with a cough during the day or night. In addition, for females only, self-reported smoking had a significant association with current wheezing and also with the occurrence of a chest cough during the most recent respiratory infection. For more details of this analysis see (24), included in the Appendix.

Table 5.1.2 gives mean values of height-adjusted pulmonary function in Period IV separately for smokers and non-smokers. There is a highly significant difference between the smoking and non-smoking groups particularly with respect to age; the smokers being on the whole approximately one year older than the non-smokers. This difference in age accounts for the differences in height and weight, and may also account for the difference in FVC, since pulmonary function was adjusted only for height, and for some variables of pulmonary function age and weight may also be important in a manner separate from their association with height. This possibility will be tested by further analyses.

5.2 Factors present **before** initiation of smoking.

This part of the analysis seeks to answer the first two research questions specified in section 1 of this report. The **outcome measure** used in each analysis was a pulmonary function variable measured **during Period IV** of this study. Since this was a continuously distributed variable, multiple regression analyses were used. The effect of a number of **independent variables measured in Period II** were then assessed. These were:

- a) The presence or absence of a number of the subject's respiratory symptoms,
- b) The presence or absence of maternal cough or smoking,
- c) The occurrence or non-occurrence of early

hospitalization of the subject for respiratory illness,

d) The level of family income.

e) Exposure to ambient (outdoor) total suspended particulates estimated as annual arithmetic mean of monthly geometric mean.

f) Exposure to the "Fine Fraction" of ambient suspended particles estimated as annual arithmetic mean of monthly geometric means.

g) Exposure to ambient SO₂ estimated as annual arithmetic mean of weekly arithmetic means.

Note: Included in the analysis with each of the above was the presence or absence of self-reported smoking during Period IV. In addition, in each analysis, to allow for baseline differences in pulmonary function (which may or may not reflect pre-existing pulmonary function abnormality), the estimate in period 2 of the particular pulmonary function variable being used as the outcome measure was included as an independent variable in the multiple regression analysis. We then examined the effects of the most important variables when introduced together in a multiple regression equation.

5.2.1 First Stage.

We performed an initial analysis to reduce the number of variables being considered to a reasonable level, because of the large number of variables examined and also because of the high degree of correlation between some of them. In this first part of the analysis therefore, each of the variables listed above was examined separately except that self-smoking in Period IV and the Period II pulmonary function variable were always introduced as independent variables. All 3 variables were introduced together, and then a stepwise analysis was performed such that a variable that failed to meet the criterion for being retained was dropped, and the analysis continued until no further variable was dropped. The analyses were performed separately for males and females for Period II and Period III.

The results of these analyses are shown on Tables 5.2.1 to 5.2.4. These tables give an overall view of those variables which, when considered separately, have a significant association with either a reduction or an increase in a particular pulmonary function variable. The association with the level of air pollutants is not shown on these tables since this was to be included in the final analysis regardless of the presence or absence of any significant association in these initial analyses.

It can be seen by the number of "0's" that the presence of a particular symptom in males in Period II had relatively little

effect on Period IV pulmonary function, although there was a significant ($p < 0.01$) reduction of FEV1/FVC ratio if the mother had been smoking during Period II. There was also a significant lengthening in MET associated with the presence of persistent wheeze. Both wheezing and day or night cough in females in Period II had a significant effect on a number of pulmonary function variables measured in Period IV (particularly MET). A positive association was seen between maternal smoking and both FEV1 and FVC. Early hospitalization for respiratory illness was not found to have any consistent effect on Period IV pulmonary function for either males or females.

In Period III, the presence of asthma in males became an important determinant of Period IV pulmonary function with a highly significant ($p < 0.005$) reduction in both FEV1/FVC and peak flow, and to a lesser extent, also in a number of other variables. The significant reduction in FEV1/FVC ratio in the presence of maternal smoking using Period III data was still evident. For females the effect of cough and wheezing was much greater than that found with Period II data.

Self-reported smoking showed a positive association with both FEV1 and FVC when measured in Period IV but only in males and not in females.

5.2.2 Second Stage.

For the second part of the analysis we selected maternal smoking, day or night cough and wheezing as the most consistently important variables seen in these initial analyses. These three variables were then introduced in stepwise fashion along with levels of air pollution and the presence or absence of reported smoking in further multiple regression analyses that are shown in Tables 5.2.5 to 5.2.12. These tables show the standardized estimated beta coefficient for each variable and the 't' value associated with that estimated beta coefficient. These analyses measure the effect of each independent variable on the particular dependent pulmonary function variable, when the other independent variables are also in the equations. The objective was to judge the relative strengths of the effects.

For Period II data the major effects seen are:

- a) FEV1/FVC is significantly reduced in females in the presence of wheezing and in males in the presence of maternal smoking.
- b) FEF75/85 also shows the significant reduction in females in the presence of wheezing.
- c) Maternal smoking in females is associated with an increase in FEV1, FVC, MEF75, FEF2575 and FEF7585.
- d) An increase in suspended particulates is associated

with an increase in FVC in females, and with increases in FEV1, FVC, MEF50, and FEF2575 in males.

e) No effect of self-reported smoking was seen in females but a positive association similar to but less than that with TSP was seen in males between the presence of self-reported smoking and with FEV1 and FVC, and also with MEF75 and FEF7585.

f) Exposure to Fine Fraction in Period II was associated with highly significant increases in FEV1, FVC, MEF50, and FEF2575 in males, and a smaller and less significant increase in FVC in females.

g) Exposure to SO2 in Period II was associated with highly significant increases in FEV1, FVC, MEF50 and FEF2575 in males, and a smaller and less significant increase in FVC in females.

For Period III data the following relationships were noted:

a) In both males and females, wheezing was associated with a reduction in FEV1, FEV1/FVC and FEF2575, and an increase in MET. In females, it was also associated with a reduction in MEF50, MEF75, and FEF7585.

b) Maternal smoking in males was still associated with a reduction in FEV1/FVC, and in females was still associated with increases in various pulmonary function variables.

c) The positive association between particulates and pulmonary function found in Period II was less marked in Period III. In females, a significant lengthening in MET was found, but in males a significant positive association with FEV1 and FVC was observed which was smaller in magnitude than in Period II.

d) The associations noted between self-reported smoking in males and females and pulmonary function observed in Period II are relatively unchanged in Period III.

5.3 Concurrent Factors

The third research question was concerned with the effect of concurrent environmental factors that might interact with cigarette smoking.

5.3.1 Stage 1.

Tables 5.3.1 - 5.3.2 show the results of analyses that were conducted in a similar manner for Period IV data to those conducted for Periods II and III data, shown in Tables 5.2.1 to 5.2.4. The individual questionnaire variables have changed

somewhat due to a change of format of the self-administered questionnaire. Self-reported smoking is taken from the same questionnaire as that for the analyses for the two previous periods. Maternal smoking is now defined by that reported by the subject in the smoking questionnaire.

The major effect seen in males is that of asthma which was associated with a significant reduction in virtually all the pulmonary function variables. This association was seen for a history of current asthma and was also seen even with only a history of asthma at some previous time. In contrast to this a history of wheezing had a lesser effect with only one significant association.

For females, there were fewer significant relationships of the individual variables noted. The presence of wheezing was associated with a reduction in FEV1/FVC and an increase in MET, but the level of significance was much less marked than when wheezing was present in previous periods.

We were also interested in examining any interaction between individual symptoms and smoking. In males, this interaction variable was found to be significant in the relationship between: morning cough, current asthma, and chest coughs with colds; and a variety of pulmonary function variables. Only in the case of current asthma and peak flow were the direct effects and the interactive effects both significant. For females there was evidence of a similar interaction of smoking with both wheezing and cough, even though only for the case of morning cough and MEF50 were both the direct and interactive effects significant.

5.3.2. Stage 2.

We then performed a multiple regression analysis for males and females separately in a manner similar to that for the first two research questions. As in these former analyses, we selected maternal smoking, day or night cough, and wheezing as the variables of interest along with self reported smoking. In addition, the level of suspended particulates was introduced as an independent variable in one set of multiple regression analyses and in the second set the fine fraction of the suspended particulates was used as the independent variable. Tables 5.3.3 and 5.3.4 show the results when total suspended particulates was used as the measure of particulate exposure and 5.3.5 and 5.3.6 the results when the fine fraction was used as a measure of particulate exposure.

For males the major association seen was that of self-reported smoking with an increase in a number of pulmonary function variables, that is: FEV1, FVC, MEF75, and FEF7505. A positive association was also seen between total suspended particulates and several pulmonary function variables, and the same was found for fine fraction. Negative associations were seen between the

presence of wheeze and both FEV1 and FEV1/FVC.

In females, self-reported smoking was associated with a reduction in FEV1/FVC and peak flow. Maternal smoking was associated with positive changes in FVC, FEV1, MEF75, and FEF2575, but the presence of a wheeze was associated with a reduction in FEV1/FVC, MEF50, FEF2575, and an increase in MET. The presence of a day or night cough was associated with a reduction in MEF75. The level of total suspended particulates was associated with an increase in MET, and no associations were found with fine fraction.

6. CONCLUSION

In this study, over a two year period, we have obtained pulmonary function data on a cohort of Canadian adolescents. Simultaneously, estimates have been obtained of their smoking habits. The validity and repeatability of these data have been estimated and are found to be within acceptable limits. Concurrently with the pulmonary function study of the children, estimates of the exposure to suspended particulates have been obtained and are reported. (Data on sulphur dioxide levels have also been obtained and will be available at a future date).

We could find little evidence that exposure, either before or during this study, to an increased level of suspended particles (even when expressed as the fine fraction) was associated with any decrement in pulmonary function. Indeed, we found that there was a positive association between the levels of suspended particles as well as fine fraction and a number of pulmonary function variables in males. Only for MET was there observed a significant association with TSP (but not FF) in females, and this positive association is interpreted as an increase in airflow obstruction.

A positive association was observed between self-reported smoking and pulmonary function but only for males. The interpretation of these data is difficult but is suggestive of some element of pre-selection. Unpublished data from our previous study indicates that those who initiate smoking in this time period had larger lung volumes before the start of smoking at the age of eight than those who have not commenced smoking. These differences were significant only for females. It may well be that in this period when smoking is starting, the opposing effects of a selection factor and of smoking itself may make the finding of any smoking effect difficult.

Of the variables which we have measured in these subjects before the start of smoking, the presence of wheezing appeared to be the one which most consistently was associated with decrement in pulmonary function during the period of this current study. This was especially true for females and in this respect, the finding of a significant interaction between wheezing and smoking in females is of importance. Whether the presence of

this particular symptom can be used to identify those at risk of pulmonary function impairment on the initiation of smoking can only be determined however by further followup of this cohort. The presence of a cough in females may be an additional risk factor.

We also found that the presence of maternal smoking in males prior to this period of study was associated with decrement in the FEV1/FVC ratio. This was not found in females and the relationship in males was not seen for maternal smoking reported during the current period of study. These data are consistent with the relationships we have described in our previous study (21).

"Small airways disease"

Since the research questions of this study centered on the issue of the development of "small airways disease" as an early feature of chronic lung disease, it was necessary to include in our measurements of pulmonary function some tests that have been accepted as indicators of the patency of small airways. These tests of "small airway function" are also influenced to a greater or less degree by other factors, which contributes to the variability of the measure. Tests of this type that we carried out are: MEF50, FEF2575, MET, MEF75, and FEF7585. The latter two (MEF75 and FEF7585) are measures of flow at the lowest lung volumes, and in theory are likely to be most influenced by small airway calibre.

It can be seen from Table 4.1.10 that there is a large and very significant difference in the values of these tests which depends on gender, and thus it is necessary to examine the results of these two tests separately in males and in females.

Self-smoking in Period IV was associated with an increase in the magnitude of these variables in males, but this may be related to the greater mean age of the smoking cohort, in spite of the fact that the pulmonary function variables were adjusted for height. This association was not found in females.

Maternal smoking in Periods II, III and IV was not associated with a change in these variables in males. In females, in Period II there was a positive association with both, and in Periods III and IV there was a positive association with MEF75.

TSP exposure in Periods II and III was not associated with any change in these variables, but exposure in Period IV was associated with a significant increase in MEF75 in males. No effects of TSP in any period were seen in females.

Fine fraction in Period IV was associated with an increase in both variables in males, but not in females.

SO₂ exposure in Period II was not associated with any changes in these variables in males or females.

Day or night cough was not associated with a change in these variables in any Period in males, but in females, in Period IV only, there was an association with a decrement in MEF75.

Wheeze in males was not associated with a change in these variables in any Period, but in females, in Periods II and III FEF7585 showed a negative association, as did MEF75. In females, as in males, this was not found with the Period IV data.

We interpret these findings to indicate that at ambient levels of TSP, fine fraction, or SO₂ measured over the course of this study, we have no evidence of a decrement in "small airways function" associated with these indicators of air pollution. In females only, decrement in "small airways function" was associated with wheeze in earlier years (Periods II and III), and with current (Period IV) cough. These latter associations were not found in males. Interactive effects in Period IV were found in males; between smoking and cough, and small airways function; and in females between smoking and wheeze, and small airways function.

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TABLE 4.1.1

AGE DISTRIBUTION OF COHORT

AGE	GENDER		TOTALS
	MALE	FEMALE	
10	0	1	1
11	127	136	263
12	205	179	384
13	464	457	921
14	150	148	298
15	85	76	161
16	27	20	47
TOTALS	1058	1017	2075

NOTES: Age is in years.
 Missing observations = 15
 (Missing subjects are those for whom
 consent was given, but testing was
 not performed due to unavailability of
 subject.)

TABLE 4.1.2
DIFFERENCES BETWEEN SUBJECTS STUDIED IN CURRENT STUDY
AND THOSE LOST TO FOLLOW-UP SINCE 1979

CHARACTERISTIC	PREVALENCE (%)		p-VALUE
	CURRENT COHORT	LOST TO FOLLOW-UP	
MALE	50.1	53.4	0.059
SUBJECT COUGH IN MORNING	5.5	7.0	0.006
SUBJECT EVER WHEEZE	24.9	26.5	0.30
MOTHER COUGH IN MORNING	14.3	21.1	<0.0001
MOTHER SMOKE	39.6	49.6	<0.0001
FATHER COUGH	25.6	26.9	0.44
FATHER SMOKE	44.2	46.1	0.26
INCOME < 10K\$	11.2	23.3	<0.0001
SERIOUS CHEST ILLNESS BEFORE AGE 2	5.9	10.3	<0.0001

Data for comparison have been taken from Period II (1979) data set.

TABLE 4.1.3

COMPARISON BETWEEN "NOT AVAILABLE" AND "AVAILABLE" GROUPS

VARIABLE MNEMONIC OR EXPLANATION	NOT AVAILABLE	PULMONARY FUNCTION DATA				CLINICALLY IMPORTANT DIFFERENCE	CID EXCEEDED
		AVAILABLE	P-VALUE	SIG.			
AGE (<10 YEARS) \$	44.1	50.3	<.0001	*		10.1	
AGE (<10 YEARS) \$	72.7	82.2	<.0001	*		16.4	
WEIGHT (kg)	34.4	33.6	.0048			3.4	
HEIGHT (cm)	138.0	137.8	.4542			13.8	
MALE \$	53.4	50.1	.0599			10.0	
FVC (ℓ)	2.32	2.31	.6236			0.23	
FEV1 (ℓ)	1.87	1.86	.4207			0.19	
FEF7585 (ℓ/sec)	0.78	0.76	.0642			0.08	
PF (ℓ/sec)	3.73	3.75	.3857			0.38	
MEF50 (ℓ/sec)	2.43	2.41	.3076			0.24	
MEF75 (ℓ/sec)	1.06	1.03	.0509			0.10	
MET (sec)	0.59	0.59	.2656			0.06	

\$ = Prevalence Rates (%)

. . . continued

TABLE 4.1.3
COMPARISON BETWEEN "NOT AVAILABLE" AND "AVAILABLE" GROUPS

VARIABLE MNEMONIC OR EXPLANATION	PULMONARY FUNCTION DATA					CLINICALLY IMPORTANT DIFFERENCE	CID EXCEEDED
	NOT AVAILABLE	AVAILABLE	P-VALUE	SIG.			
VC (ℓ)	2.33	2.93	.6849			0.23	
RV (ℓ)	0.61	0.62	.1757			0.06	
FRC (ℓ)	1.31	1.32	.4659			0.13	
CV (ℓ)	0.27	0.28	.2909			0.03	
N2DIFF (%/ℓ)	0.92	0.94	.4813			0.09	
IDI	1.68	1.66	.0477			0.17	

TABLE 4.1.4
SMOKING STATUS BY AGE

<u>SMOKING CATEGORY</u>					
n (%prevalence)*					
AGE	NEVER	EXPERIMENTAL	1-10/WEEK	>10/WEEK	TOTAL
10	1	0	0	0	1
11	196(76.6)	56(21.9)	4(1.6)	0	256
12	227(63.2)	114(31.8)	17(4.7)	1(0.3)	359
13	480(53.6)	324(36.2)	61(6.8)	30(3.4)	895
14	141(48.6)	107(36.9)	21(7.2)	21(7.2)	290
15	59(37.6)	58(36.9)	17(10.8)	23(14.6)	157
16	12(27.3)	16(36.4)	6(13.6)	10(22.7)	44
TOTALS	1116(55.7)	675(33.7)	126(6.3)	85(4.2)	2002

NOTES: Age is in years.

Missing observations = 88

*:% prevalence is the percentage based on the
row total, rounded to one decimal place.

TABLE 4.1.5
DIFFERENCE IN CARBON MONOXIDE LEVEL BY SMOKING CATEGORY

SMOKING CATEGORY	NUMBER	CO DIFFERENCE* (mean, ppm)	STANDARD DEVIATION
NEVER SMOKED	1116	2.22	1.77
EXPERIMENTAL	659	2.55	2.13
1-10 CIG/WK	125	4.35	4.27
>10 CIG/WK	85	9.78	7.87
TOTAL	1985		

*Difference in ppm between carbon monoxide concentrations in expired air and in ambient air.

Missing cases = 105

TABLE 4.1.6
RELATIONSHIP BETWEEN MATERNAL AND PERSONAL SMOKING

	MOTHER SMOKING		TOTAL
	YES	NO	
0*	637(83.8)**	1101(93.0)	1738
PERSONAL SMOKING			
1-10/WK	70(9.2)	51(4.3)	121
>10/WK	52(7.0)	32(2.7)	84
TOTAL	759	1184	1943

NOTES: * Includes experimental smoking
 ** Column percentage

Missing observations = 149 147
 $\chi^2 = 40.60$ $p < 0.001$

Entries in core of table are of form:
 n (prevalence %)
 "Total" entries are of form: n

TABLE 4.1.7
REPRODUCIBILITY OF QUESTIONNAIRE DATA

QUESTION	n	MISSING	RAW AGREEMENT	CHANCE-CORRECTED AGREEMENT (K)
COUGH IN MORNING	146	4	0.95	0.81
COUGH DAY OR NIGHT	146	4	0.90	0.70
CHEST EVER SOUND WHEEZY	146	4	0.90	0.73
PREVIOUS HISTORY OF ASTHMA	146	4	1.0	1.0
COLD USUALLY ACCOMPANIED BY COUGH	146	4	0.93	0.78
GAS COOKING	146	4	0.98	0.93
HISTORY OF EVERY SMOKING	141	9	0.99	0.97
HISTORY OF CURRENT SMOKING	62*	3	0.95	0.87
MOTHER SMOKE	138	12	1.0	1.0
OTHERS IN HOUSE SMOKE				

*This question only answered by those with a positive response to the previous question (ever smoked).

TABLE 4.1.8

REPRODUCIBILITY OF PULMONARY FUNCTION DATA

VARIABLE*	n	INITIAL TEST** (MEAN, SD)	REPEAT TEST** (MEAN, SD)	t-VALUE ⁺	p-VALUE
FVC	150	3.36, 0.75	3.38, 0.75	-1.85	0.07
FEV1	150	2.73, 0.61	2.74, 0.60	-0.31	0.76
$\dot{V}_{50}$	150	3.45, 0.98	3.46, 0.96	-0.47	0.64
$\dot{V}_{75}$	150	1.64, 0.63	1.62, 0.64	1.01	0.31
MET	150	0.59, 0.17	0.59, 0.18	-0.87	0.38
PF	150	5.29, 1.26	5.30, 1.11	-0.11	0.91

* See glossary for description of abbreviations

** Values rounded to two decimal places

+ Significance of difference between variable means in smoking and non-smoking groups.

TABLE 4.1.9

PREVALENCE OF RESPIRATORY SYMPTOMS BY GENDER

SYMPTOM	MALE (n=1047), %	FEMALE (n=1008), %	X , P**	TOTAL (n=2055), %
MORNING COUGH*	141, 13.5	160, 15.9	2.38, >0.1	301, 14.6
MORNING COUGH FOR 3 MONTHS	24, 2.3	37, 3.7	3.42, 0.061	61, 3.0
DAY OR NIGHT COUGH	206, 19.7	248, 24.6	7.25, 0.007	454, 22.1
DAY OR NIGHT COUGH FOR 3 MONTHS	39, 3.7	59, 5.9	5.12, 0.022	98, 4.8
EVER WHEEZE	216, 20.6	203, 20.1	0.15, >0.1	419, 20.4
CURRENT WHEEZE	61, 5.8	56, 5.6	0.07, >0.1	117, 5.7
EVER ASTHMA	106, 10.1	70, 6.9	6.63, 0.010	176, 8.6
CURRENT ASTHMA	63, 6.0	45, 4.5	2.49, >0.1	108, 5.3
CHEST COUGH WITH ANY COLD	417, 39.8	429, 42.6	1.58, >0.1	846, 41.2
CHEST COUGH WITH LAST COLD	316, 30.2	295, 29.3	0.09, >0.1	611, 29.7

* Detail of question given in Kerigan (1).

** Significance of difference in prevalence between genders.
Missing Cases - 35

TABLE 4.1.10

MEAN VALUES OF PULMONARY FUNCTION IN PERIOD IV

VARIABLE	MALES		SIG	FEMALES	
	MEAN	SD		MEAN	SD
AGE (yr)	13.44	1.15		13.39	1.14
HEIGHT (cm)	158.82	10.65	****	156.71	7.73
WEIGHT (kg)	50.71	13.19		49.79	11.68
FVC (l)	3.52	.80	****	3.18	.57
FEV1 (l)	2.80	.65	****	2.66	.48
FEV1/FVC (per cent)	79.80	6.80	****	83.90	6.70
FEF25-75 (l/s)	2.98	.91	***	3.13	.82
FEF75-85 (l/s)	1.19	.52	****	1.36	.49
PF (l/s)	5.45	1.30	****	5.13	1.05
MEF50 (l/s)	3.43	1.03	*	3.56	.91
MEF75 (l/s)	1.57	.62	****	1.74	.60
MET (s)	.637	.307	****	.541	.214

Note: p value bounds given by:

* = $p < 0.01$ *** = $p < 0.0001$ **** = $p < 0.00001$

Table 4.2.1

UAEG TSP $\mu\text{g}/\text{m}^3$

MONTHLY GEOMETRIC MEANS 1982

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	102	-	38	-	-	-	-	-	44	41	32	36	30
UP. OTTAWA	104	59	35	53	42	61	43	44	31	34	30	31	29
ABERDEEN	106	76	38	71	53	123	67	58	37	36	36	38	31
RED HILL	109	70	38	57	46	58	43	52	33	31	31	31	34
HIGHVIEW	110	-	-	69	51	85	54	59	44	45	44	47	40
CATHEDRAL	112	60	46	81	63	126	82	68	46	61	54	50	53
McELROY 2	113	-	47	87	54	155	82	66	45	47	42	44	36
WESTMOUNT	114	77	40	63	41	100	57	52	33	35	33	37	31
BISHOPSGT	115	73	40	64	55	-	39	52	35	38	35	35	31
DUNDURN C	116	55	41	70	49	74	52	61	41	43	38	36	44
M.O.E.	117	82	48	84	67	70	50	70	56	57	46	44	40
WOODWARD	118	70	54	80	72	73	64	73	60	62	66	51	60
WHITNEY 2	119	68	44	72	53	94	60	57	34	36	32	38	43
SAN DIEGO	120	53	34	59	42	91	58	64	35	38	33	35	24
MT. ALBION	121	54	50	79	47	84	64	67	43	46	39	41	40
ALBERTON	122	62	31	48	44	83	49	51	28	36	29	26	27
U. WENTWTH	123	64	39	75	55	109	65	59	40	46	42	46	37

MOE TSP ($\mu\text{g}/\text{m}^3$)

HUGH/HUNT	301	59	44	96	60	157	77	96	45	60	52	49	79
CITY HALL	302	55	32	75	37	150	95	84	44	59	48	68	52
NORTHPARK	303	112	113	89	106	58	44	78	80	80	93	69	79
KEN'WORTH	304	75	64	105	60	102	79	72	36	45	46	47	51
BURL/LEED	305	51	82	142	105	264	132	108	76	93	92	64	49
BURL/WELL	306	100	53	106	64	113	72	79	81	70	63	59	54
CHAT/FRID	307	74	52	146	77	157	100	100	54	65	66	70	81
BART/WENT	308	61	63	86	82	194	110	97	88	101	72	85	60
HUGHSON N	309	77	48	71	42	92	54	60	39	57	58	39	36
UWELL/INV	310	47	47	85	67	144	75	63	43	39	44	44	38
CUMBERLND	311	55	31	82	66	139	76	59	35	52	48	52	47
BART/NASH	312	54	42	102	71	93	88	62	52	68	44	40	46
WESTDL LB	313	60	54	103	-	92	57	57	44	49	43	46	41

Table 4.2.2

UAEG TSP ug/m3

MONTHLY GEOMETRIC MEANS 1983

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	102	35	35	54	43	57	66	52	58	50	29	51	43
UP. OTTAWA	104	36	35	42	36	41	53	36	46	35	26	42	48
ABERDEEN	106	41	42	57	38	61	71	37	49	-	-	-	-
RED HILL	109	41	36	42	33	42	55	39	41	47	26	45	38
HIGHVIEW	110	49	52	65	68	57	72	48	63	46	38	55	47
CATHEDRAL	112	54	59	72	68	62	81	47	75	-	-	-	-
McELROY 2	113	39	45	65	51	58	69	44	60	41	35	50	41
WESTMOUNT	114	37	37	63	51	48	58	36	53	36	27	44	37
BISHOPSGT	115	43	37	51	60	47	58	43	51	38	32	47	45
DUNDURN C	116	49	41	40	31	52	64	47	51	45	36	49	32
M.O.E.	117	-	51	-	-	58	78	56	64	51	29	56	47
WOODWARD	118	58	58	44	48	60	87	62	68	64	-	53	56
WHITNEY 2	119	44	41	48	32	50	54	42	43	35	27	32	37
SAN DIEGO	120	38	29	45	33	53	69	37	48	46	29	48	39
MT. ALBION	121	50	44	63	33	64	72	44	54	51	-	-	43
ALBERTON	122	29	27	36	31	44	53	33	49	36	32	41	34
U. WENTWTH	123	51	54	76	-	58	67	53	57	-	-	-	-

MOE TSP (ug/m3)

HUGH/HUNT	301	61	53	83	65	85	95	52	78	53	48	54	46
CITY HALL	302	59	69	60	58	88	88	-	-	-	-	-	-
NORTH PARK	303	79	70	69	46	77	107	111	59	95	56	103	124
KEN' WORTH	304	50	64	60	62	63	69	47	65	41	44	54	52
BURL/LEED	305	94	79	107	137	116	112	63	115	74	86	109	62
BURL/WELL	306	56	62	59	66	104	89	60	72	67	55	70	-
CHAT/FRID	307	73	60	65	58	119	99	71	83	80	81	90	90
BART/WENT	308	70	64	122	78	118	116	71	110	69	64	87	76
HUGHSON N	309	41	43	43	39	65	75	44	48	48	30	49	45
UWELL/INV	310	40	50	64	64	79	80	42	54	43	39	50	51
CUMBERLND	311	47	60	79	62	67	71	52	72	73	46	59	52
BART/NASH	312	61	-	46	38	64	62	43	64	65	82	70	69
WESTDL LB	313	29	30	51	41	58	58	55	51	72	50	64	51

Table 4.2.3

UAEg TSP ug/m3

MONTHLY GEOMETRIC MEANS 1984

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	102	33	56	44	37	-	81	-	130	44	66	-	50
UP. OTTAWA	104	37	40	37	38	40	52	35	59	32	40	31	32
RED HILL	109	39	46	37	58	38	49	49	59	30	31	38	31
HIGHVIEW	110	51	59	50	69	64	68	58	68	37	49	39	43
McELROY 2	113	43	57	40	76	51	83	58	69	37	55	41	44
WESTMOUNT	114	39	43	39	73	39	55	45	55	34	52	35	38
BISHOPSGT	115	40	50	45	70	50	64	57	64	34	43	34	38
DUNDURN C	116	43	58	36	48	46	51	57	72	37	52	52	41
M.O.E.	117	55	68	-	120	61	62	66	86	47	53	53	44
WOODWARD	118	63	75	45	91	77	69	78	95	58	53	67	43
WHITNEY 2	119	43	45	32	59	49	56	48	63	29	44	39	41
SN DIEGO2	120	42	58	41	67	47	-	49	59	33	-	-	-
MT ALBION	121	52	-	52	-	29	-	-	-	32	-	-	13
ALBERTON	122	29	35	32	52	39	54	-	-	25	44	17	27
BART/WENT	124	64	105	67	119	82	94	78	90	59	106	66	61
SN DIEGO3	125	-	-	-	-	-	-	-	-	-	50	33	26

MOE TSP (ug/m3)

HUGH/HUNT	301	68	86	55	69	97	103	81	97	62	79	70	65
NORTHPARK	303	131	104	72	83	112	49	102	130	63	72	83	121
KEN'WORTH	304	52	62	61	101	66	72	55	82	38	61	58	57
BURL/LEED	305	77	116	103	161	109	141	86	98	72	105	107	88
BURL/WELL	306	59	71	55	79	93	97	95	128	73	74	84	94
CHAT/FRID	307	72	84	51	100	114	111	80	102	71	97	128	72
BART/WENT	308	75	109	63	107	103	105	74	88	54	118	71	72
HUGHSON N	309	44	38	60	74	71	75	63	65	48	59	46	58
UWELL/INV	310	49	66	47	71	58	9	52	63	34	53	43	51
CUMBERLND	311	50	68	49	75	55	66	57	75	44	63	63	-
BART/NASH	312	41	53	41	-	70	71	77	82	45	56	64	52
WESTDL LB	313	71	95	64	64	70	69	64	65	39	58	73	66
BAY/MAIN	318	43	61	41	54	67	93	46	79	43	68	53	42

Table 4.2.5

ANNUAL GEOMETRIC MEANS 1982-1985

UAEG TSP ug/m3

SITE NAME	NUMBER	EASTING	NORTHING	1982	1983	1984	1985
MT. HOPE	102	0	0	37	46	52	45
UP. OTTAWA	104	2910	1240	39	39	39	36
ABERDEEN	106	1548	2545	51	49	-	-
RED HILL	109	4312	1864	42	40	42	37
HIGHVIEW	110	3060	1860	53	54	54	45
CATHEDRAL	112	2535	2610	63	65	-	-
McELROY 2	113	1986	1912	56	49	52	48
WESTMOUNT	114	1230	1830	45	44	45	39
BISHOPSGT	115	2396	1496	45	45	47	41
DUNDURN C	116	1665	3285	49	43	49	41
M.O.E.	117	4945	1945	57	54	61	52
WOODWARD	118	4622	2720	64	58	67	52
WHITNEY 2	119	441	2709	50	39	44	38
SAN DIEGO	120	630	2278	43	42	45	-
MT. ALBION	121	4030	1450	51	50	36	38
ALBERTON	122	-	-	39	37	34	35
U. WENTWTH	123	2379	2079	53	59	-	-
BART/WENT	124	2739	2895	74	78	79	67
SN DIEGO3	125	645	2283	-	-	-	41

MOE TSP (ug/m3)

HUGH/HUNT	301	2050	2715	67	64	76	55
CITY HALL	302	1950	2770	60	68	-	-
NORTHPARK	303	4225	3773	80	73	89	-
KEN' WORTH	304	3670	2355	62	55	67	56
BURL/LEED	305	3250	3045	92	95	113	113
BURL/WELL	306	2480	3350	73	68	73	83
CHAT/FRID	307	1410	2805	81	80	84	61
BART/WENT	308	2739	2895	81	75	85	71
HUGHSON N	309	2285	3310	54	47	59	52
UWELL/INV	310	2140	2245	57	53	59	58
CUMBERLND	311	2968	2278	58	61	59	54
BART/NASH	312	4745	2185	60	57	55	55
WESTDL LB	313	1148	3037	53	50	71	47
BAY/MAIN	318	1900	2795	-	-	-	39
BEACH BLV	315	4426	3486	-	-	-	69

Table 4.2.6

UAEG PSD 1982

MONTHLY MEAN; MASS MEDIAN DIAMETER (μm)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	201	-	1.89	1.65	3.42	2.84	2.98	3.26	2.81	3.03	-	2.63	2.33
BART/WENT	202	2.91	2.34	3.02	3.79	3.41	3.46	3.75	5.13	4.58	3.34	3.71	3.21
NORTHPARK	203	3.77	2.79	2.86	5.45	3.38	3.25	3.67	4.87	2.93	4.44	4.31	3.27
SENECA	204	3.06	1.26	1.45	2.83	2.84	2.94	2.52	2.24	2.56	-	-	1.75
RED HILL	205	2.66	1.34	.87	2.81	2.83	2.71	2.53	1.98	-	-	-	-
HIGHVIEW	207	-	-	2.14	3.02	3.09	2.61	3.39	1.69	2.65	-	2.58	2.18
WHITNEY 2	208	2.61	1.46	1.38	2.44	2.46	2.75	2.64	1.85	2.59	-	1.18	1.57
SAN DIEGO	209	-	1.86	2.23	2.79	2.33	2.32	2.46	2.13	-	-	-	-
U.OTTAWA	210	2.99	1.36	1.17	3.21	3.03	2.13	2.68	1.63	-	-	-	-

Table 4.2.7

UAEG PSD 1983

MONTHLY MEAN; MASS MEDIAN DIAMETER (μm)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	201	2.13	2.95	2.94	2.68	3.86	3.01	3.81	3.27	3.37	-	3.13	4.88
BART/WENT	202	3.06	3.84	-	3.82	4.53	4.18	3.57	2.95	4.67	3.63	3.76	4.47
NORTHPARK	203	3.09	4.24	-	3.56	4.72	4.18	3.46	3.48	3.98	6.65	2.46	3.33
SENECA	204	1.85	2.48	3.01	2.91	3.21	3.12	3.28	3.04	3.06	3.41	2.1	2.43
RED HILL	205	-	-	-	-	2.93	3.15	2.92	2.71	.97	4.21	2.18	3.15
HIGHVIEW	207	2.3	2.78	-	2.88	3.31	3.31	3.12	3.02	3.69	3.34	2.38	2.99
WHITNEY 2	208	2.08	1.83	2.61	2.37	2.97	2.86	3.05	2.38	2.02	3.14	1.85	2.54
SAN DIEGO	209	-	-	-	-	2.96	2.67	3.39	-	2.81	3.26	1.86	3.24
U.OTTAWA	210	-	-	-	-	2.94	3.15	2.97	3.29	2.87	2.46	1.81	2.71

Table 4.2.8

UAEG PSD 1984

MONTHLY MEAN; MASS MEDIAN DIAMETER (μm)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	201	2.84	3.11	5.37	3.57	-	4.05	-	4.91	4.66	3.46	4.03	4.04
BART/WENT	202	3.33	4.29	3.63	4.27	3.22	4.65	2.72	2.78	3.45	3.71	5.03	4.41
NORTHPARK	203	4.04	5.59	4.14	-	4.59	3.58	4.08	3.19	3.91	3.29	5.86	-
SENECA	204	2.42	2.83	3.67	3.38	2.95	3.35	2.79	2.31	2.78	3.18	2.51	3.27
RED HILL	205	2.26	2.93	3.11	3.48	2.72	3.08	1.97	1.21	2.57	3.09	2.03	2.44
HIGHVIEW	207	3.12	4.61	3.14	3.15	3.72	3.61	2.32	1.98	2.84	2.58	3.11	2.96
WHITNEY 2	208	2.01	3.03	3.18	3.46	2.84	3.47	2.18	2.05	1.84	2.59	2.3	2.7
SAN DIEGO	209	2.78	2.83	3.61	3.39	2.97	2.69	2.44	1.85	1.51	-	-	-
U.OTTAWA	210	2.42	3.02	3.41	3.79	2.44	3.12	2.48	1.64	1.9	3.08	1.49	1.07
SANDIEGO3	211	-	-	-	-	-	-	-	-	-	2.56	3.35	2.9
BEACH BLV	212	-	-	-	-	-	-	-	-	-	-	-	5.05

Table 4.2.9

UAEG PSD 1985

MONTHLY MEAN; MASS MEDIAN DIAMETER (μm)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	201	-	2.55	3.92	4.65	5.68	3.97	3.97	3.18	3.41	3.57	2.64	3.25
BART/WENT	202	2.93	3.47	5.15	4.45	4.35	4.12	4.11	3.46	4.25	2.47	3.11	2.99
SENECA	204	2.79	1.6	2.1	3.28	4.5	3.45	3.47	2.98	2.55	2.06	2.33	2.8
RED HILL	205	2.69	2.42	2.25	1.93	4.08	2.81	1.8	2.28	3.01	1.21	1.89	1.82
HIGHVIEW	207	2.94	2.36	3.67	3.1	1.94	3.01	3.16	2.86	3.02	-	2.61	2.13
WHITNEY 2	208	2.79	2.05	3.21	2.21	4.17	3.21	2.65	1.94	2.26	2	1.85	1.22
U.OTTAWA	210	2.09	2.17	2.09	2.63	4	2.67	2.67	1.89	2.17	1.59	1.18	1.58
SANDIEGO3	211	2.48	2.08	3.78	2.75	4.53	3.01	3.2	2.68	3.12	1.88	3.13	2.82
BEACH BLV	212	3.45	4.43	4.93	5.27	4.42	4.42	5.22	4.1	7.17	4.99	2.05	4.93

Table 4.2.10

UAEG PSD 1982

MONTHLY MEAN; FINE FRACTION & COARSE FRACTION ($\mu\text{g}/\text{m}^3$)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	201 FF	-	44	62	54	85	69	49	37	38	-	51	35
	CF	-	30	38	58	75	57	48	34	31	-	28	31
BART/WENT	202	131	74	82	71	125	116	107	66	67	68	87	45
		110	55	86	78	132	116	112	104	81	66	89	60
NORTHPARK	203	114	85	81	89	57	57	67	52	62	70	70	57
		138	78	78	125	59	57	75	70	62	89	82	67
SENECA	204	78	50	65	53	103	84	65	35	42	-	-	33
		80	22	42	45	81	68	50	26	30	-	-	23
RED HILL	205	-	-	-	54	67	59	62	36	-	-	-	-
		57	28	23	46	57	44	46	24	-	-	-	-
HIGHVIEW	207	-	-	67	62	87	65	67	33	40	-	55	43
		-	-	47	56	84	53	68	24	34	-	42	34
WHITNEY 2	208	75	59	83	58	95	67	75	37	32	-	58	44
		69	29	53	47	72	55	55	24	26	-	31	23
SAN DIEGO	209	-	50	77	46	90	73	64	35	-	-	-	-
		-	27	55	41	62	52	49	22	-	-	-	-
U.OTTAWA	210	59	46	53	53	76	72	57	28	-	-	-	-
		58	20	26	51	76	44	44	18	-	-	-	-

Table 4.2.11

UAEG PSD 1983

MONTHLY MEAN; FINE FRACTION & COARSE FRACTION ($\mu\text{g}/\text{m}^3$)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	201 FF	46	32	75	74	78	95	46	64	147	-	47	39
	CF	28	33	63	65	92	82	51	60	54	-	42	70
BART/WENT	202	52	49	-	83	65	97	52	100	40	54	64	51
		48	59	-	91	85	119	55	88	50	55	72	69
NORTHPARK	203	74	40	-	38	57	90	91	55	88	76	89	103
		60	47	-	43	70	106	-	54	99	110	74	119
SENECA	204	52	33	89	46	51	66	51	57	27	29	48	50
		27	29	72	38	49	59	44	49	26	30	32	50
RED HILL	205	-	-	-	-	45	65	49	52	70	28	45	29
		-	-	-	-	40	57	40	44	26	30	29	32
HIGHVIEW	207	46	51	-	55	52	68	53	71	36	45	55	48
		35	45	-	50	54	66	49	58	33	45	39	47
WHITNEY 2	208	50	39	57	32	52	62	44	56	41	31	42	46
		29	23	45	24	46	51	38	37	29	26	27	36
SAN DIEGO	209	-	-	-	-	50	69	40	-	29	41	47	40
		-	-	-	-	47	53	40	-	24	38	27	44
U.OTTAWA	210	-	-	-	-	47	61	42	46	28	31	41	36
		-	-	-	-	42	55	36	39	22	25	24	33

Table 4.2.12

UAEG PSD 1984

MONTHLY MEAN; FINE FRACTION & COARSE FRACTION ($\mu\text{g}/\text{m}^3$)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	201 FF	47	54	55	48	-	59	-	84	40	57	41	62
	CF	41	51	88	50	-	74	-	149	56	64	38	84
BART/WENT	202	55	81	85	128	86	66	72	65	45	77	47	44
		52	96	94	153	84	85	61	58	49	86	52	57
NORTHPARK	203	77	93	61	326	75	70	78	79	48	58	39	-
		100	131	83	248	94	78	93	78	57	64	62	-
SENECA	204	42	69	72	98	48	60	61	70	32	53	36	36
		33	54	63	119	39	60	56	47	28	51	28	37
RED HILL	205	47	56	56	65	34	38	51	51	34	34	26	28
		33	47	52	81	29	39	28	23	27	33	19	25
HIGHVIEW	207	49	67	99	94	45	58	57	69	39	58	40	52
		45	114	82	108	45	58	45	42	36	41	33	47
WHITNEY 2	208	75	64	54	99	52	56	59	62	33	52	51	40
		39	57	47	116	44	59	37	38	23	35	34	33
SAN DIEGO	209	43	95	47	87	46	57	55	51	27	-	-	-
		35	64	50	101	40	46	39	31	16	-	-	-
U.OTTAWA	210	46	58	63	73	45	49	38	60	30	37	28	31
		30	52	74	101	32	46	30	31	21	35	18	17

Table 4.2.13

UAEG PSD 1985

MONTHLY MEAN; FINE FRACTION & COARSE FRACTION ($\mu\text{g}/\text{m}^3$)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MT. HOPE	201 FF	-	44	51	51	57	44	45	56	50	42	25	68
	CF	-	33	62	59	102	50	58	55	51	43	22	64
BART/WENT	202	58	51	48	68	66	56	67	65	42	44	31	33
		51	51	71	85	88	68	84	68	57	40	34	36
SENECA	204	47	46	29	46	49	47	44	42	39	25	22	44
		36	28	24	46	66	46	48	39	33	20	17	48
RED HILL	205	42	34	32	32	38	35	28	56	27	31	20	31
		30	25	25	25	43	29	26	35	32	14	14	24
HIGHVIEW	207	56	64	35	50	75	42	44	46	38	-	34	44
		46	47	43	47	88	40	44	43	37	-	28	34
WHITNEY 2	208	55	44	56	37	55	41	45	40	28	22	26	27
		52	26	50	29	74	35	38	23	24	17	20	17
U.OTTAWA	210	43	43	26	32	33	37	38	40	28	21	27	34
		26	27	20	25	63	29	32	26	22	15	11	22
SN DIEGO3	211	38	37	32	44	44	40	44	41	33	25	21	31
		30	21	39	39	59	36	46	34	32	18	31	28
BEACH BVD	212	61	51	70	58	73	62	41	49	54	36	21	70
		74	69	100	78	96	85	59	59	90	53	16	98

Table 4.2.15

UAEG 802 1980

MONTHLY ARITHMETIC MEAN (ppb)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MACASSA LODGE	401	-	-	-	-	-	-	-	-	-	-	-	-
CHEDOKE HOSP	402	-	-	-	-	-	6.7	5.5	-	7.2	5.6	-	5
MACDONALD HS	403	-	-	-	-	-	-	-	-	-	-	-	-
DUNDURN CASTLE	404	-	-	-	-	-	-	-	-	-	-	-	-
WESTMOUNT HS	405	20.2	8.1	7.1	-	-	-	10.1	7.3	10.4	2.5	7	0
CHURCHILL HS	406	-	-	-	-	-	-	-	-	-	-	-	-
SHERWOOD HS	407	-	-	-	-	-	-	-	-	-	-	-	-
MCMASTER UNIV	408	3.5	9.1	9.7	11.7	7.2	-	9.1	6.4	-	-	9.2	5.8
CENTENNIAL PS	409	22.6	-	12.1	9.4	-	12	9.8	-	5.6	3.9	-	14.5
SOUTHMOUNT HS	410	3.2	-	7.9	12.3	-	11.6	10.5	-	3.9	5.3	-	10.7
HENDERSON HOSP	411	3	-	11.4	7	-	11.8	9.7	-	12.9	14.5	-	7.8
DELTA HS	412	14.3	-	12.7	8.8	-	-	-	-	-	-	-	-
RIDGEMOUNT PS	413	2.8	-	6.9	9.1	-	16.7	14.5	-	9.1	6.4	-	-
GLENDALE HS	414	26.3	-	19	13	-	5.1	6	-	9.8	10.2	-	22.1
TWEEDSMUIR PS	415	-	9.3	19.9	11	19.8	12.1	14	14.8	18	10	17	11.4
ST JOSEPH HOSP	416	9.7	-	6.6	7	-	22.1	13.6	-	7.2	13.4	-	15.6
WOODWARD PS	417	1.1	12.1	11.4	5.1	5.7	0	9.9	7.2	5.7	6.7	8.6	-
ROSEDALE PS	418	-	10.8	31.5	7.5	3	2.5	2.6	3.5	3.3	3.8	6.6	4.1
BRANTDALE PS	419	-	-	-	-	11.8	-	4.6	6	-	2.2	7.9	6.8
HILL PARK HS	420	2.3	9.3	9.9	12.7	7	.8	11.6	6.9	2	12.2	8.2	6.2
LISGAR PS	421	10.8	7.9	13.6	10.1	9	1.7	.9	4.27	5.2	4.6	6	4
BART/WENT OME	422	15.5	14.9	19	22.2	13.4	14	8.2	8.7	11.5	10	9.4	11.2
NORTHPARK OME	423	13.1	13.2	10	17.7	3.7	10.4	3.4	6	12.7	18.6	22.2	31.1
KING GEORGE PS	424	-	-	-	-	-	-	9.2	-	6	8.9	-	0

Table 4.2.16

UAEG SO2 1981

MONTHLY ARITHMETIC MEAN (ppb)

SITE NAME	NUMBER	JAN	FEB	MAR	APR	MA	JUN	JUL	AUG	SEP	OCT	NOV	DEC
MACASSA LODGE	401	-	-	-	-	-	-	-	-	-	-	-	-
CHEDOKE HOSP	402	16.8	-	5.7	5.7	-	5.5	13.8	-	14.1	7.9	-	-
MACDONALD HS	403	-	-	-	-	-	-	-	-	-	-	-	-
DUNDURN CASTLE	404	-	-	-	-	-	-	-	-	-	-	-	-
WESTMOUNT HS	405	4.6	11.2	2.6	15.2	23.9	29.7	6.9	9.6	5.9	2.8	4	-
CHURCHILL HS	406	-	-	-	-	-	-	-	-	-	-	-	-
SHERWOOD HS	407	-	-	-	-	-	-	-	-	-	-	-	-
MCMASTER UNIV	408	9.4	7.9	2.8	6.2	8.9	7.9	8.6	7	-	10.6	11	7.7
CENTENNIAL PS	409	16.5	-	4.4	4.7	-	7.8	9.1	-	9.8	5.9	-	-
SOUTHMOUNT HS	410	19.1	-	4.5	-	-	7.1	11.9	-	6.5	6.3	-	-
HENDERSON HOSP	411	8.3	-	9.3	5.3	-	237	37.2	-	12.3	13.9	-	-
DELTA HS	412	-	-	-	-	-	-	-	-	-	-	-	-
RIDGEMOUNT PS	413	7.1	-	4.2	4	-	16	7.8	14.6	6.9	10.9	-	-
GLENDALE HS	414	13.8	-	8.1	11.8	-	21.7	-	-	8.9	3.7	-	-
TWEEDSMUIR PS	415	10.9	6.7	-	7.8	14.6	21.9	16.4	18.5	18.2	22	14.9	8.5
ST JOSEPH HOSP	416	18.2	-	4.6	6.6	-	5.5	6.9	-	7.7	15.1	-	-
WOODWARD PS	417	3.8	8.1	.3	4	5.5	-	46.8	19.5	20.5	27.8	10.9	8.9
ROSEDALE PS	418	16.9	12.5	24	5.3	9.4	3.4	5.7	6.5	12.5	6.5	7	7.2
BRANTDALE PS	419	21.2	35.8	-	5.6	8.3	28	3.6	6.6	6.9	3.7	10.5	4.2
HILL PARK HS	420	7.6	15	2.7	7	10.2	20	5.8	7.6	-	12.9	5.7	3.1
LISSAR PS	421	11.3	12.2	4	7.4	7.4	6	9.7	8.3	-	9.4	7.9	9.7
BART/WENT OME	422	15	17.1	10.3	13	16.5	10	7.7	6.1	4	8.1	10.3	6.5
NORTHPARK OME	423	29.6	169.9	17.4	116.9	12.9	76.3	67.3	10.3	7	6.1	8	14.2
KING GEORGE PS	424	10.2	-	7.3	9.6	-	8.5	12.5	-	11.3	-	-	-

Table 4.2.17

PERIOD IV SO2 NETWORK VALID DATA PER SITE

SITE NAME	SITE #	START	END	OP DAYS
FERNWOOD N.	451	83-04-21	85-02-21	516
EAST 12th	452	83-03-30	85-03-10	606
MAIN WEST	454	83-07-04	83-07-21	17
HUNTER & HESS	455	83-11-17	85-03-10	479
WESTDALE	456	83-12-02	84-09-28	295
MAIN EAST	457	84-01-05	85-03-10	405
SENECA	458	84-01-23	85-03-10	355
FERNWOOD S.	459	84-11-13	85-03-10	94
PARADISE	460	85-02-08	85-03-10	30
NEWLANDS	461	85-02-20	85-03-10	18

(MISSING CASES - 102 (100 for last quarter))

* Translator reduced by 1.

TABLE 5.1.1

PREVALENCE OF RESPIRATORY SYMPTOMS BY
ADOLESCENT SMOKING BEHAVIOUR

SMOKING BEHAVIOUR

SYMPTOM	<u>non-smoker</u> (n=1775), %	<u>1-10/wk.</u> (n=125), %	<u>>10/wk.</u> (n=85), %	<u>TOTAL</u> (n=1985), %
MORNING COUGH*	239, 13.5	26, 20.8	25, 29.4	290, 14.6
MORNING COUGH FOR 3 MONTHS	44, 2.5	4, 3.2	11, 12.9	59, 3.0
DAY OR NIGHT COUGH	357, 20.1	43, 34.4	37, 43.5	437, 22.0
DAY OR NIGHT COUGH FOR 3 MONTHS	74, 4.2	8, 6.4	12, 14.1	94, 4.7
EVER WHEEZE	347, 19.5	24, 19.2	30, 35.3	401, 20.2
CURRENT WHEEZE	87, 4.9	8, 6.4	15, 17.6	110, 5.5
EVER ASTHMA	151, 8.5	9, 7.2	5, 5.9	165, 8.3
CURRENT ASTHMA	94, 5.3	5, 4.0	2, 2.4	101, 5.1
CHEST COUGH WITH ANY COLD	713, 40.2	59, 47.2	46, 54.1	818, 41.2
CHEST COUGH WITH LAST COLD	*511, 28.8	46, 36.8	34, 40.0	*591, 29.8

MISSING CASES - 105 (106 for last question)

* Denominator reduced by 1.

Table 5.1.2

MEAN VALUES OF HEIGHT-ADJUSTED PULMONARY FUNCTION IN PERIOD IV
BY SMOKING CATEGORY

VARIABLE	MALES					FEMALES				
	nonsmoking		smoking			non-smoking		smoking		
	MEAN	SD	MEAN	SD		MEAN	SD	MEAN	SD	
AGE (yr)	13.37	1.12	14.26	1.15	***	13.27	1.09	14.11	1.13	***
HEIGHT (cm)	158.41	10.66	164.32	9.17	***	156.45	7.97	158.60	6.15	**
WEIGHT (kg)	49.74	12.08	55.92	13.00	***	48.95	10.99	52.08	9.59	**
FVC (l)	3.51	.41	3.64	.47	*	3.16	.37	3.28	.41	***
FEV1 (l)	2.79	.36	2.90	.44		2.65	.32	2.72	.37	
FEV1/FVC (%)	79.81	6.76	79.79	6.80		83.97	6.61	82.95	7.47	
FEF25-75 (l/s)	2.97	.72	3.11	.84		3.11	.70	3.20	.80	
FEF75-85 (l/s)	1.19	.42	1.29	.53		1.35	.44	1.70	.48	
PF (l/s)	5.46	.94	5.31	1.07		5.14	.90	5.08	.95	
MEF50 (l/s)	3.42	.83	3.58	.95		3.53	.80	3.65	.89	
MEF75 (l/s)	1.56	.51	1.67	.60		1.73	.54	1.77	.60	
MET (s)	.6341	.2797	.6758	.5624		.5416	.2248	.5472	.1505	

Note: p value bounds given by:

* = $p < 0.01$ ** = $p < 0.001$ *** = $p < 0.0001$

NOTES ON TABLES 5.2.1 TO 5.2.4

A stepwise regression process was used to obtain these tables as follows:

The dependent variable (ie. the variable to be predicted) is a Period IV lung function variable (e.g.FVC) which appears as one of the column headings in each table. The corresponding Period II lung function variable, the Period IV self-reported smoking variable, and **one** of the Period II or III independent variables in the far left column of each table were all initially forced into the simple linear regression model.

The F-test significance level used for the deletion of an independent variable was a probability of .1 and the significance level used for the addition of an independent variable back into the regression model was a probability of .05.

The underlined +s, -s and 0s in these tables correspond to variables remaining in the model at the completion of the stepwise regression process. The 0s not underlined indicate that the corresponding independent variable was **not** in the model at the completion of the stepwise regression process. Further, had this variable been included, its coefficient's t-value would have corresponded to a probability, p, greater than .05.

Please note that each column contains a summary of twelve separate regression processes (ie. one for each of the twelve independent variables other than the Period II lung function variable and the Period IV self-reported smoking variable). The probability level indicated for the t-value of the self-reported smoking variable's coefficient is a general estimated level intended to be representative overall of the results of the twelve stepwise regression processes involved.

The questionnaire variables (apart from the income variable) were coded "1" for the presence of the symptom or characteristic and "0" for its absence. The income variable was coded "1" for a family income greater than or equal to \$10,000 and "0" for less than \$10,000. Also, the following lung function variables have been adjusted for height for boys and girls separately in both Periods II and IV: FEV1, FVC, MEF50, MEF75, PF, FEF2575 and FEF7585.

5.2.1

MULTIPLE REGRESSION ANALYSIS

(stage 1)

INDEPENDENT VARIABLES FROM PERIOD II (EXCEPT WHERE NOTED)

MALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

	FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. II									
Morning cough	0	0	0	0	0	---	0	0	0
Morning cough for 3 months	0	0	0	0	0	0	0	0	0
Day/night cough	0	0	0	0	0	---	0	0	±
Day/night cough for 3 months	0	0	0	0	0	0	0	0	0
Wheeze Ever	0	0	0	0	0	0	0	0	0
Persistent wheeze	0	±	0	0	0	0	0	0	++++
Asthma	0	0	0	0	0	0	0	0	0
Cold going to chest	0	0	0	0	0	0	0	0	0
Mother smoke	0	0	---	0	0	0	0	0	0
Mother cough	0	--	0	0	0	0	0	0	0
Early hosp. for RTI	0	0	0	0	0	0	0	0	0
Income	±	0	0	0	±	0	0	0	0
Per. IV s.r. smoking	++	++	0	0	+++	0	0	++	0

increases with variable present

decreases with variable present

+ p < 0.05

- p < 0.05

++ p < 0.02

-- p < 0.02

+++ p < 0.01

--- p < 0.01

++++ p < 0.005

---- p < 0.005

Variable not significant

"-----" underlining means variable in the model

0 p ≥ 0.05

Period IV pulmonary function as dependent variable.

Period IV self-smoking and Period II pulmonary function as independent variables.

Questionnaire variables considered individually.

For further detail, see p. 53.

5.2.2

MULTIPLE REGRESSION ANALYSIS
(stage 1)
INDEPENDENT VARIABLES FROM PERIOD II (EXCEPT WHERE NOTED)
FEMALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

	FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. II									
Morning cough	0	0	0	=	--	0	=	=	++++
Morning cough for 3 months	0	0	0	0	--	0	<u>0</u>	---	±
Day/night cough	0	0	<u>0</u>	<u>0</u>	<u>0</u>	0	--	0	++++
Day/night cough for 3 months	0	0	0	0	0	0	<u>0</u>	0	0
Wheeze Ever	0	0	----	0	0	0	0	=	++
Persistent wheeze	--	<u>0</u>	0	--	=	0	=	=	±
Asthma	0	0	0	0	0	0	0	0	0
Chest illness in last 12 months	0	0	0	0	0	0	0	0	0
Mother smoke	++++	+++	0	<u>0</u>	+++	0	±	±	0
Mother cough	0	<u>0</u>	<u>0</u>	0	0	<u>0</u>	=	0	<u>0</u>
Early hosp. for RTI	0	0	0	0	0	0	0	0	0
Income	0	0	0	0	0	0	0	0	0
Per. IV s.r. smoking	0	0	<u>0</u>	0	0	<u>0</u>	0	0	0

increases with variable present

decreases with variable present

+ p < 0.05

- p < 0.05

++ p < 0.02

-- p < 0.02

+++ p < 0.01

--- p < 0.01

++++ p < 0.005

---- p < 0.005

Variable not significant

"____"

underlining means variable in the model

0 p ≥ 0.05

Period IV pulmonary function as dependent variable.

Period IV self-smoking and Period II pulmonary function as independent variables.

Questionnaire variables considered individually.

For further detail, see p. 53.

5.2.3

MULTIPLE REGRESSION ANALYSIS

(stage 1)

INDEPENDENT VARIABLES FROM PERIOD III (EXCEPT WHERE NOTED)

MALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

	FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. III									
Morning cough	0	0	0	0	0	0	0	0	0
Morning cough for 3 months	0	0	0	<u>0</u>	0	0	0	0	0
Day/night cough	0	0	0	0	0	-	0	0	0
Day/night cough for 3 months	0	0	0	0	-	0	0	0	0
Wheeze ever	<u>0</u>	0	--	<u>0</u>	<u>0</u>	-	<u>0</u>	<u>0</u>	±
Persistent wheeze	0	0	0	0	<u>0</u>	0	0	0	0
Asthma	--	0	----	---	--	----	---	--	<u>0</u>
Chest illness in last 12 months	0	0	0	0	0	-	0	0	0
Mother smoke	0	+	--	0	0	0	0	0	0
Mother cough	0	--	0	<u>0</u>	0	0	0	0	0
Early hosp. for RTI	0	0	<u>0</u>	--	0	<u>0</u>	<u>0</u>	0	<u>0</u>
Income	<u>0</u>	0	0	0	<u>0</u>	0	0	0	0
Per. IV s.r. smoking	++	++	0	0	++	0	<u>0</u>	±	0

increases with variable present

decreases with variable present

+ p < 0.05

- p < 0.05

++ p < 0.02

-- p < 0.02

+++ p < 0.01

--- p < 0.01

++++ p < 0.005

---- p < 0.005

Variable not Significant

0 p ≥ 0.05

"_____" underlining means variable in the model

Period IV pulmonary function as dependent variable.

Period IV self-smoking and Period II pulmonary function as independent variables.

Questionnaire variables considered individually.

For further detail, see p. 53.

5.2.4

MULTIPLE REGRESSION ANALYSIS
(stage 1)
INDEPENDENT VARIABLES FROM PERIOD III (EXCEPT WHERE NOTED)
FEMALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

	FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. III									
Morning cough	<u>-</u>	<u>0</u>	0	0	0	<u>0</u>	0	0	0
Morning cough for 3 months	<u>-</u>	0	0	0	0	0	0	0	0
Day/night cough	<u>-</u>	0	<u>---</u>	<u>0</u>	<u>-</u>	<u>--</u>	<u>-</u>	0	0
Day/night cough for 3 months	<u>----</u>	<u>0</u>	<u>---</u>	<u>-</u>	<u>----</u>	<u>----</u>	<u>--</u>	<u>---</u>	0
Wheeze	<u>--</u>	0	<u>----</u>	<u>--</u>	<u>--</u>	0	<u>---</u>	<u>-</u>	<u>++</u>
Persistent wheeze	0	0	0	0	0	0	0	0	<u>0</u>
Asthma	0	0	0	0	<u>0</u>	<u>0</u>	0	0	0
Chest illness in last 12 months	0	0	0	0	0	0	0	0	0
Mother smoke	<u>++</u>	<u>+++</u>	0	0	<u>++</u>	0	<u>0</u>	<u>+</u>	0
Mother cough	0	<u>0</u>	<u>0</u>	0	0	0	0	0	<u>0</u>
Early hosp. for RTI	<u>+</u>	<u>+</u>	0	0	0	0	0	0	0
Income	0	0	0	0	0	0	0	0	0
Per. IV s.r. smoking	0	0	<u>-</u>	0	0	0	0	0	0

increases with variable present

decreases with variable present

+ p < 0.05

- p < 0.05

++ p < 0.02

-- p < 0.02

+++ p < 0.01

--- p < 0.01

++++ p < 0.005

---- p < 0.005

Variable Not Significant

"_____" underlining means variable in model

0 p > 0.05

Period IV pulmonary function as dependent variable.

Period IV self-smoking and Period II pulmonary function as independent variables.

Questionnaire variables considered individually.

For further detail, see p. 53 .

NOTES ON TABLES 5.2.5 - 5.2.12; and 5.3.3 - 5.3.6

A stepwise regression process was used to obtain these tables as follows:

The dependent variable (ie. the variable to be predicted) is a Period IV lung function variable (e.g.FVC) which appears as one of the column headings in each table. The independent variables in the far left column were all initially forced into the simple linear regression model.

The F-test significance level used for the deletion of an independent variable was a probability of .1 and the significance level used for the addition of an independent variable back into the regression model was a probability of .05. The R^2 's listed in the tables have been adjusted for degrees of freedom.

Please note that in the above table:

1) 'beta-hat' is the estimated regression coefficient standardized with respect to the corresponding independent variable's mean and standard deviation

2) 'beta-hat' is, also, the partial correlation between the corresponding independent variable and the Period IV lung function variable, taking into account the rest of the variables in the regression model. Thus 'beta-hat' is between -1 and 1 inclusively.

3) with the standardized coefficients, the constant term equals 0.

The 'beta-hats' underlined in the tables correspond to variables remaining in the model at the completion of the stepwise process. The 'beta-hats' not underlined in the tables correspond to the variables not in the model at the completion of the stepwise process. Also, the 'beta-hats' not underlined are the 'beta-hats' that would occur if the corresponding variable had been entered on the next step. The 'beta-hats' in the model can be compared directly for the relative effects of the corresponding independent variables.

The questionnaire variables were coded "1" for the presence of the symptom and "0" for its absence. The air quality variables, TSP and PF (micrograms/cubic metre), and SO₂ (parts per billion) are yearly mean exposures. The following lung function variables have been adjusted for height for boys and girls separately in both Periods II and IV: FEV₁, FVC, MEF₅₀, MEF₇₅, PF, PEF₂₅₇₅ and PEF₇₅₈₅.

5.2.5

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD II
MALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. II										
Day or										
night	β	-0.018	-0.003	-0.021	-0.026	-0.019	<u>-0.062</u>	-0.026	-0.004	<u>0.03899</u>
cough	t	-0.77	-0.143	-0.99	-1.22	-0.81	-2.29*	-1.27	-0.162	2.042*
Wheeze	β	0.010	0.012	0.011	-0.025	0.001	-0.019	-0.006	0.008	-0.00958
	t	0.43	0.59	0.51	-1.14	0.05	-0.70	-0.28	0.34	-0.490
Mother	β	-0.008	0.033	<u>-0.058</u>	0.008	-0.007	-0.029	-0.006	-0.0077	-0.01264
smoke	t	-0.33	1.53	-2.67***	0.37	-0.30	-1.08	-0.26	-0.31	-0.664
TSP	β	<u>0.080</u>	<u>0.069</u>	0.024	<u>0.048</u>	<u>0.045</u>	0.034	<u>0.043</u>	0.04	-0.01980
	t	3.45****	3.25****	1.10	2.23*	1.95	1.25	2.08*	1.62	-1.042
Per. IV										
s.r.	β	<u>0.052</u>	<u>0.046</u>	0.020	0.031	<u>0.058</u>	-0.033	<u>0.036</u>	<u>0.60</u>	-0.02632
smoking	t	2.26*	2.167*	0.90	1.46	2.50**	-1.23	1.74	2.38**	-1.387
Per. II	β	<u>.68622</u>	<u>.74028</u>	<u>.73149</u>	<u>.74032</u>	<u>.68520</u>	<u>.53172</u>	<u>.75862</u>	<u>.61874</u>	<u>.79871</u>
Pulmonary	t	29.769 [#]	34.958 [#]	33.858 [#]	34.535 [#]	29.566 [#]	19.744 [#]	36.577 [#]	24.756 [#]	41.826 [#]
Function										
Adjusted R ²		.47815	.56047	.54211	.54799	.47172	.28939	.57691	.38531	.64582

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variable.

Stepwise deletion of selected questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.2.6

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD II
FEMALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. II										
Day or										
night	β	-0.021	-0.007	-0.024	<u>-0.04554</u>	<u>-0.03930</u>	0.002	<u>-0.05165</u>	-0.010	<u>0.03899</u>
cough	t	-0.92	-0.372	-0.99	-1.98*	-1.73	0.075	-2.39**	-0.40	2.042*
Wheeze										
	β	<u>-0.03909</u>	0.018	<u>-0.08940</u>	-0.028	-0.026	-0.038	-0.019	<u>-0.057</u>	-0.00958
	t	-1.73	0.92	-3.75****	-1.17	-1.11	-1.32	-0.86	-2.30**	-0.490
Mother										
smoke	β	<u>0.06899</u>	<u>0.051</u>	0.023	<u>0.04354</u>	<u>0.05995</u>	-0.008	<u>0.05009</u>	<u>0.058</u>	-0.01264
	t	3.06****	2.56**	0.98	1.90	2.63***	-0.28	2.32**	2.37**	-0.664
TSP										
	β	0.023	<u>0.055</u>	-0.019	-0.013	0.015	0.021	-0.007	0.01	-0.01980
	t	1.02	2.76***	-0.81	-0.57	0.65	0.72	-0.31	0.42	-1.042
Per. IV										
s.r.	β	-0.013	0.003	<u>-0.04543</u>	0.005	-0.019	<u>-0.04871</u>	-0.015	-0.013	-0.02632
smoking	t	-0.55	0.15	-1.923	0.216	-0.83	-1.71	-0.67	-0.53	-1.387
Per. II	β	<u>.70619</u>	<u>.77955</u>	<u>.66125</u>	<u>.69563</u>	<u>.70665</u>	<u>.46922</u>	<u>.73695</u>	<u>.64300</u>	<u>.79871</u>
Pulmonary	t	31.267 [#]	39.146 [#]	27.749 [#]	30.171 [#]	31.024 [#]	16.485 [#]	34.057 [#]	26.104 [#]	41.826 [#]
Function										
Adjusted R ²		.50968	.61818	.45991	.48974	.49949	.21830	.54880	.42151	.64582

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variable.

Stepwise deletion of selected questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.2.7

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD III
MALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. III										
Day or										
night	β	-0.0011	-.01285	0.00108	-0.00237	-0.00368	<u>-.05126</u>	-0.00120	.00405	.00886
cough	t	-0.046	-.597	0.048	-0.106	-0.152	-1.848	-0.056	.155	.450
Wheeze										
	β	<u>-0.04613</u>	-.01795	<u>-.05043</u>	<u>-0.04200</u>	<u>-0.04456</u>	<u>-.04680</u>	<u>-0.04312</u>	<u>-.04953</u>	<u>.04066</u>
	t	-1.971*	-.831	-2.29*	-1.926	-1.881	-1.687	-2.040*	-1.943	2.103*
Mother										
	β	0.00600	<u>.04037</u>	<u>-.05137</u>	0.00459	0.01339	-.01916	0.00393	.000835	-.00858
Smoke	t	0.252	1.851	-2.34**	0.210	0.565	-.706	0.186	.033	-.446
TSP										
	β	<u>0.05369</u>	<u>.04824</u>	0.01501	0.01842	0.02420	.00257	0.01440	.02312	-.01399
	t	2.291*	2.220*	0.679	0.846	1.024	.095	0.683	.908	-.729
Per. IV										
s.r.	β	<u>0.05917</u>	<u>.04823</u>	0.01997	<u>.03852</u>	<u>0.06649</u>	-.03118	<u>.04393</u>	<u>.06497</u>	-.02753
smoking	t	2.53**	2.238*	0.911	1.774	2.819***	-1.153	2.088*	2.558**	-1.436
Per. II										
Pulmonary	β	<u>.67921</u>	<u>.73440</u>	<u>.72554</u>	<u>.73335</u>	<u>.67436</u>	<u>.54201</u>	<u>.75121</u>	<u>.60743</u>	<u>.79793</u>
Function	t	29.015 [#]	34.028 [#]	33.028 [#]	33.635 [#]	28.467 [#]	20.047 [#]	35.543 [#]	23.832 [#]	41.266 [#]
Adjusted R ²		.47336	.55674	.54053	.54631	.46462	.29648	.57392	.37896	.64607

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variable.

Stepwise deletion of selected questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.2.8

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD III
FEMALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. III										
Day or										
night	β	<u>-.04439</u>	-.01582	<u>-.05317</u>	-.03671	<u>-.04411</u>	<u>-.07053</u>	<u>-.03737</u>	-.02138	.02459
cough	t	-1.891	-.782	-2.144*	-1.517	-1.861	-2.441**	-1.650	-.832	1.040
Wheeze	β	<u>-.05070</u>	-.00301	<u>-.06909</u>	<u>-.06028</u>	<u>-.04859</u>	-.02349	<u>-.05252</u>	<u>-.05570</u>	<u>.06179</u>
	t	-2.157*	-.149	-2.783****	-2.530**	-2.042*	-.796	-2.306*	-2.205*	2.648***
Mother	β	<u>.04607</u>	<u>.04756</u>	-.00371	.01013	<u>.04887</u>	-.03094	.02524	<u>.04464</u>	-.00719
smoke	t	2.007*	2.348**	-0.151	0.429	2.104*	-1.072	1.142	1.773	-.312
TSP	β	-0.01867	.02027	<u>-.04352</u>	-.03152	-.00899	<u>-.05102</u>	-.02819	-.02608	<u>.05147</u>
	t	-0.808	.999	-1.785	-1.333	-0.386	-1.772	-1.272	-1.032	2.243*
Per. IV										
s.r.	β	-.00869	.00894	<u>-.04273</u>	.01595	-.01790	-.03949	-0.00236	-.01106	.02188
smoking	t	-0.372	.435	-1.755	0.675	-0.762	-1.358	-0.107	-.435	.946
Per. II										
Pulmonary	β	<u>.70426</u>	<u>.78289</u>	<u>.65560</u>	<u>.68389</u>	<u>.69973</u>	<u>.46561</u>	<u>.72494</u>	<u>.63812</u>	<u>.70172</u>
Function	t	30.618 [#]	38.660 [#]	26.816 [#]	28.702 [#]	29.948 [#]	16.124 [#]	32.393 [#]	25.186 [#]	30.052 [#]
Adjusted R ²		.51018	.61995	.45728	.48241	.50096	.22845	.54587	.41348	.51053

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variable.

Stepwise deletion of selected questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.2.9
 MULTIPLE REGRESSION ANALYSIS
 (stage 2)
 INDEPENDENT VARIABLES FROM PERIOD II
 MALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. II										
Day or	β	-.01546	-.00295	-.02175	-.02491	-.01739	-.06228	-.02531	-.00405	.03899
night	t	.671	-.139	-1.004	-1.159	-.748	-2.316*	-1.217	-.162	2.042*
cough										
Wheeze	β	.01422	.01265	.01161	-.02184	.00353	-.01862	-.00353	.00848	-.00958
	t	.618	.595	.532	-1.017	.152	-.674	-.170	.339	-.490
Mother	β	-.00413	.03591	-.05962	.00970	-.00435	-.03210	-.00369	-.00773	-.01264
Smoke	t	-.179	1.688	-2.758***	.452	-.186	-1.193	-.177	-.307	-.664
FF	β	.09269	.06750	.03759	.06293	.04211	.05327	.05362	.04115	-.02696
	t	4.024*****	3.190****	1.737	2.938****	1.815	1.973*	2.585***	1.646	-1.421
Per. IV										
s.r.	β	.05174	.4304	.01859	.03080	.05817	-.03479	.03577	.05938	-.02632
smoking	t	2.249*	2.028*	.859	1.439	2.508**	-1.296	1.726	2.376**	-1.387
Per. II	β	.68937	.73965	.73415	.74236	.68560	.53755	.76000	.61874	.79871
Pulmonary	t	29.932 [#]	34.897 [#]	33.931 [#]	34.661 [#]	29.568 [#]	19.870 [#]	36.664 [#]	24.756 [#]	41.826 [#]
Function										
Adjusted R ²		.48039	.56140	.54305	.54967	.47145	.29148	.57792	.38531	.64582

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variable.

Period IV stepwise deletion of selected questionnaire and air quality variables.

Self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.2.10

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD II
FEMALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. II										
Day or	β	-.02147	-.00826	-.02421	-.04554	-.03930	.00215	-.05165	-.01020	.06010
night	t	.923	-.415	-.990	-1.976*	-1.728	.075	-2.388**	-.404	2.599***
cough										
Wheeze	β	-.03909	.01903	-.08940	-.02797	-.02620	-.03773	-.01929	-.05659	.04006
	t	-1.728	.955	-3.752****	-1.167	-1.106	-1.320	-.855	-2.298*	1.714
Mother	β	.06899	.05426	.02338	.04354	.05995	-.00803	.05009	.05812	-.03369
Smoke	t	3.060****	2.722***	.977	1.896	2.632***	.280	2.319*	2.371**	-1.505
FF	β	.03611	.05016	.00352	.00429	.03412	.04090	.01247	.02753	.01261
	t	1.603	2.522**	.148	.186	1.496	1.437	.575	1.119	.560
Per. IV	β	-.01256	.00483	-.04543	.00502	-.01925	-.04871	-.01469	-.01313	.03093
S. R.	t	-.549	.239	-1.923	.216	-.836	-1.711	-.672	-.531	1.383
Smoking										
Per. II	β	.70619	.78056	.66125	.69563	.70665	.46922	.73695	.64300	.70283
Pulmonary	t	31.267 [#]	39.157 [#]	27.749 [#]	30.171 [#]	31.024 [#]	16.485 [#]	34.057 [#]	26.104 [#]	30.935 [#]
Function										
Adjusted R ²		.50968	.61770	.45991	.48974	.49949	.21830	.54880	.42151	.51617

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variables.

Period IV stepwise deletion of selected questionnaire and air quality variables.

Self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.2.11

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD II
MALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. II										
Day or										
night	β	-.01143	.714 $\times 10^{-4}$	-.02139	-.02188	-.01559	-.06016	-.02269	-.00405	.03899
cough	t	-.495	.003	-.986	-1.019	-.670	-2.236*	-1.092	-.162	2.042*
Wheeze	β	.01741	.01478	.01113	-.01936	.00492	-.01763	-.00138	.00848	-.00958
	t	.756	.695	.510	-.902	.212	-.638	-.066	.339	-.490
Mother	β	.00169	.04005	-.05758	.01346	-.00181	-.02892	-.366 $\times 10^{-3}$	-.00773	-.01264
smoke	t	.073	1.886	-2.665***	.629	-.077	-1.076	-.018	-.307	-.664
SO2	β	.08919	.07059	.02404	.07262	.03951	.05072	.06255	.02980	-.02924
	t	3.873****	3.341****	1.115	3.399****	1.703	1.888	3.021****	1.192	-1.542
Per. IV										
s.r.	β	.05125	.04197	.01960	.02994	.05794	-.03528	.03500	.05938	-.02632
smoking	t	2.226*	1.977*	.904	1.401	2.497**	-1.313	1.690	2.376**	-1.387
Per. II	β	.68682	.74076	.73149	.74076	.68438	.53335	.75855	.61874	.79871
Pulmonary	t	29.838 [#]	34.960 [#]	33.858 [#]	34.671 [#]	29.523 [#]	19.820 [#]	36.666 [#]	24.756 [#]	41.826 [#]
Function										
Adjusted R ²		.47977	.56184	.54211	.55099	.47124	.29124	.57897	.38531	.64582

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variable.

Stepwise deletion of selected questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.2.12

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD II
FEMALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. II										
Day or										
night	β	-.02147	-.00736	-.02421	-.04554	-.03930	.00215	-.05165	-.01020	.06010
cough	t	-.923	-.369	-.990	-1.976*	-1.728	.075	-2.388**	-.404	2.599***
	β	-.03909	.01972	-.08940	-.02797	-.02620	-.03773	-.01929	-.05659	.04006
Wheeze	t	-1.728	.989	-3.752****	-1.167	-1.106	-1.320	-.855	-2.298*	1.714
	β	.06899	.05760	.02338	.04354	.05995	-.00803	.05009	.05812	-.03369
Mother	t	3.060****	2.885****	.977	1.896	2.632***	.280	2.319**	2.371**	-1.505
smoke										
	β	.02896	.04154	-.01062	-.00168	.03595	.02022	.00109	.03969	.01508
SO2	t	1.283	2.086*	-.448	-.073	1.579	.711	.050	1.620	.672
Per. IV										
s.r.	β	-.01256	.00589	-.04543	.00502	-.01925	-.04871	-.01469	-.01313	.03093
	t	-.549	.291	-1.923	.216	-.836	-1.711	-.672	-.531	1.383
smoking										
Per. II	β	.70619	.77888	.66125	.69563	.70665	.46922	.73695	.64300	.70283
Pulmonary	t	31.267 [#]	39.048 [#]	27.749 [#]	30.171 [#]	31.024 [#]	16.485 [#]	34.057 [#]	26.104 [#]	30.935 [#]
Function										
Adjusted R ²		.50968	.61691	.45991	.48974	.49949	.21830	.54880	.42151	.51617

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variable.

Stepwise deletion of selected questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

NOTES ON TABLES 5.3.1 TO 5.3.2

A stepwise regression process was used to obtain these tables and is described in the following:

The dependent variable (ie. the variable to be predicted) is a Period IV lung function variable (e.g.FVC) which appears as one of the column headings in each table. The corresponding Period II lung function variable, the Period IV self-reported smoking variable, and **one** of the Period IV independent variables in the far left column of each table were all initially forced into the simple linear regression model. **Except** for the models including the maternal smoking variable, an interaction term between the included symptom variable and the Period IV self-reported smoking variable was also initially forced into the regression model.

The F-test significance level used for the deletion of an independent variable was a probability of .1 and the significance level used for the addition of an independent variable back into the regression model was a probability of .05.

The underlined +s, -s and 0s in these tables correspond to variables remaining in the model at the completion of the stepwise regression process. The 0s not underlined indicate that the corresponding independent variable was **not** in the model at the completion of the stepwise regression process. Further, had this variable been included, its coefficient's t-value would have corresponded to a probability, p, greater than .05.

Please note that each column contains a summary of eleven separate regression processes (i.e. one for each of the eleven independent variables other than the Period II lung function variable and the Period IV self-reported smoking variable). The probability level indicated for the t-value of the self-reported smoking variable's coefficient is a general estimated level intended to be representative overall of the results of the eleven stepwise regression processes involved.

The questionnaire variables were coded "1" for the presence of the symptom or characteristic and "0" for its absence. Also, the following lung function variables have been adjusted for height for boys and girls separately in both Periods II and IV: FEV1, FVC, MEF50, MEF75, PF, FEF2575 and FEF7585.

5.3.1

MULTIPLE REGRESSION ANALYSIS
(stage 1)
INDEPENDENT VARIABLES FROM PERIOD IV
MALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

	FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. IV									
Morning cough	0	0	0	0	0***	----	0*	0	0
Morning cough for 3 months	0	0	0	0	0*	0	0	0	0
Day or night cough	0	0	0	0	0	=	0	0	0
Day or night cough for 3 months	0	0	0	0	0	<u>0</u>	0	0	0
Wheeze ever	<u>0</u>	<u>0</u>	=	0	0	<u>0</u>	0	0	0
Persistent wheeze	0	0	0	0	0	0	0	0	0
Asthma ever	=	<u>0</u>	---	---	--	----	----	0	----
Current asthma	0	=	----	----	---	----**	----	----	----
Chest cough with any cold	0**	0**	0	0	0	0	0	0**	0
Chest cough with most recent cold	0*	0**	<u>0</u>	0	0	0	0	0	0*
Mother smoking	0	0	0	<u>0</u>	<u>0</u>	0	0	0	0
Per. IV s.r. smoking	++	±	0	0	±	0	<u>0</u>	++	0

increases with variable present decreases with variable present Interaction with s.r. smoking

+ p < 0.05

- p < 0.05

* p < 0.05

++ p < 0.02

-- p < 0.02

** p < 0.02

+++ p < 0.01

--- p < 0.01

*** p < 0.01

++++ p < 0.005

---- p < 0.005

variable not significant

"-----" underlining means variable in the model

0 p ≥ 0.05

Period IV pulmonary function as dependent variable.

Period IV self-smoking and Period II pulmonary function as independent variables.

Period IV questionnaire variables (other than self-smoking) considered individually.

For further detail, see p. 36.

5.3.2

MULTIPLE REGRESSION ANALYSIS
(stage 1)
INDEPENDENT VARIABLES FROM PERIOD IV
FEMALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

	FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF-25-75	FEF75-85	MET
Per. IV									
Morning cough	<u>0</u>	0*	0	<u>-</u>	<u>0</u>	0	0	0	0
Morning cough for 3 months	0	0	0	0	0	<u>-</u>	0	0	0
Day or night cough	0	0	<u>0</u>	0	<u>-</u>	<u>0</u>	0	0	0
Day or night cough for 3 months	0	0	0	0	0	0	0	0	0
Wheeze ever	0	0	<u>-</u>	0	0***	0	0***	0*	0**
Persistent wheeze	0	0	<u>-</u>	0	0	0***	0*	0	<u>++</u>
Asthma ever	0	0	0	0	0	0	0	<u>0</u>	<u>0</u>
Current asthma	0	0	0	0	0	0	0	0	<u>0</u>
Chest cough with any cold	0	0	0**	<u>0</u>	0	<u>0*</u>	<u>--</u>	0	<u>0</u>
Chest cough with most recent cold	<u>0</u>	0	0**	0	<u>-</u>	<u>-</u>	<u>0</u>	0	0*
Mother smoking	<u>+</u>	<u>+</u>	0	0	<u>+</u>	0	<u>0</u>	0	0
Per. IV s.r. smoking	0	0	<u>0</u>	0	0	0	0	0	0

increases with variable present

+ p < 0.05

++ p < 0.02

+++ p < 0.01

++++p < 0.005

decreases with variable present

- p < 0.05

-- p < 0.02

--- p < 0.01

---- p < 0.005

Interaction with S.R. Smoking

* p < 0.05

** p < 0.02

*** p < 0.01

variable not significant

0 p ≥ 0.05

"_____" underlining means variable in model

Period IV pulmonary function as dependent variable.

Period IV self-smoking and Period II pulmonary function as independent variables.

Period IV questionnaire variables (other than self-smoking) considered individually.

For further detail, see p. 67.

5.3.3

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD IV
MALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. IV										
Day or										
night	β	-.02180	-.01094	-.02390	-.01889	-.03330	-.04987	-.03150	-.02372	.01544
cough	t	-.898	-.489	-1.063	-.860	-1.391	-1.771	-1.469	-.922	.804
Wheeze	β	-.06100	-.03635	-.05105	-.01897	-.01324	-.05212	-.01058	-.00764	.00228
	t	-2.594***	-1.680	-2.304*	-.859	-.554	-1.850	-.493	-.296	.117
Mother	β	.00624	.02441	-.03376	.01197	.00356	-.00788	.00198	.00703	-.01664
Smoke	t	.263	1.111	-1.536	.545	.149	-.285	.093	.274	-.868
TSP	β	.08069	.08148	.01395	.05897	.05549	.02318	.04961	.03553	-.02233
	t	3.439****	3.763****	.634	2.703***	2.344**	.837	2.344**	1.393	-1.166
Per. IV										
s.r.	β	.05774	.04935	.01891	.03101	.05538	-.01539	.03672	.06106	-.02039
smoking	t	2.454**	2.272*	.858	1.417	2.340**	-.553	1.735	2.400**	-1.065
Per. II	β	.67969	.74031	.72871	.73894	.68269	.52195	.75719	.61943	.80786
Pulmonary	t	28.946 [#]	34.260 [#]	32.885 [#]	33.875 [#]	28.928 [#]	18.919 [#]	35.878 [#]	24.352 [#]	42.182 [#]
Function										
Adjusted R ²		.48193	.55870	.54230	.54891	.47225	.28310	.57794	.38665	.65227
*	p < 0.05									
**	p < 0.02									
***	p < 0.01									
****	p < 0.005									
#	P < 0.00005									

Period IV pulmonary function as dependent variable.

Stepwise deletion of selected questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.3.4

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD IV
FEMALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. IV										
Day or										
night	β	-.01632	.01944	-.02868	.01183	-.05055	-.03688	-.01526	-.02628	-.488x10 ⁻³
cough	t	-.706	.953	-1.143	.487	-2.191*	-1.267	-.671	-1.053	-.021
Wheeze	β	-.02779	.01230	-.05261	-.04627	-.02819	-.03916	-.04584	-.03219	.05038
	t	-1.201	.602	-2.175*	-1.980*	-1.167	-1.355	-2.087*	-1.283	2.196*
Mother	b	.05338	.04611	.01392	.03443	.05026	-.00118	.04321	.03238	-.01719
smoke	t	2.315*	2.258*	.572	1.478	2.175*	-.040	1.980*	1.297	-.756
TSP	β	-.00223	.02593	-.03281	-.03858	.00561	-.03254	-.02814	-.01519	.04590
	t	-.096	1.270	-1.364	-1.658	.242	-1.118	-1.288	-.606	2.027*
Per. IV										
s.r.	β	-.02193	.00255	-.04805	.00912	-.02129	-.05743	-.02115	-.01194	.02467
smoking	t	-.936	.124	-2.007*	.389	-.904	-1.990*	-.956	-.478	1.082
Per. II	β	.70452	.77656	.67082	.69799	.70768	.46721	.73878	.64437	.71102
Pulmonary	t	30.554 [#]	38.026 [#]	27.759 [#]	29.812 [#]	30.688 [#]	16.188 [#]	33.685 [#]	25.835 [#]	30.985 [#]
Function										
Adjusted R ²		.5000	.60994	.46241	.49301	.50128	.21761	.55405	.41459	.51832

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variable.

Stepwise deletion of selected Period IV questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.3.5

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD IV
MALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. IV										
Day or										
night	β	-.01747	-.00572	-.02390	-.01581	-.03283	-.04987	-.02989	-.02734	.01544
cough	t	-.719	-.254	-1.063	-.720	-1.378	-1.771	-1.397	-1.063	.804
Wheeze	β	-.06152	-.03715	-.05105	-.01914	-.01201	-.05212	-.01032	-.00567	.00228
	t	-2.606**	-1.707	-2.304*	-.865	-.503	-1.850	-.481	-.220	.117
Mother	β	.00875	.02863	-.03376	.01364	.933x10 ⁻⁵	-.00788	.00135	-.415x10 ⁻³	-.01664
smoke	t	.366	1.295	-1.536	.619	.000	-.285	.063	-.016	-.868
FF	β	.05292	.04077	.01950	.04107	.07756	.01271	.04907	.05955	-.01032
	t	2.249*	1.874	.887	1.879	3.288****	.460	2.320*	2.341**	-.539
Per. IV										
s.r.	β	.06058	.05309	.01891	.03282	.05443	-.01539	.03727	.05695	-.02039
smoking	t	2.567**	2.433**	.858	1.499	2.308*	-.553	1.762	2.239*	-1.065
Period II	β	.67824	.73950	.72871	.73855	.68380	.52195	.75699	.61947	.80786
Pulmonary	t	28.788 [#]	34.033 [#]	32.885 [#]	33.786 [#]	29.062 [#]	18.919 [#]	35.865 [#]	24.411 [#]	42.182 [#]
Function										
Adjusted R ²		.47824	.55374	.54230	.54712	.47518	.28310	.57789	.38954	.65227

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

[#] p < 0.00005

Period IV pulmonary function as dependent variable.

Stepwise deletion of selected Period IV questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

5.3.6

MULTIPLE REGRESSION ANALYSIS
(stage 2)
INDEPENDENT VARIABLES FROM PERIOD IV
FEMALES

PREDICTION OF PULMONARY FUNCTION IN PERIOD IV:

		FEV ₁	FVC	FEV ₁ /FVC	MEF50	MEF75	PF	FEF25-75	FEF75-85	MET
Per. IV										
Day or										
night	β	-.01632	.01944	-.02868	.00877	-.05055	-.03688	-.01526	-.02628	.488x10 ⁻⁵
cough	t	-.706	.953	-1.143	.362	-2.191*	-1.267	-.671	-1.053	.000
Wheeze	β	-.02779	.01230	-.05261	-.04736	-.02819	-.03916	-.04584	-.03219	.05179
	t	-1.201	.602	-2.175*	-2.026*	-1.167	-1.355	-2.087*	-1.283	2.257*
Mother	β	.05338	.04611	.01392	.03180	.05026	-.00118	.04321	.03238	-.01620
smoke	t	2.315*	2.258*	.572	1.366	2.175*	-.040	1.980*	1.297	-.713
FF	β	-.00136	.02484	-.02544	-.02974	.01541	-.03271	-.02024	-.110x10 ⁻³	.04181
	t	-.059	1.219	-1.055	-1.279	.665	-1.120	-.927	-.004	1.847
Per. IV										
s.r.	β	-.02193	.00255	-.04805	.00487	-.02129	-.05743	.02115	-.01194	.02426
smoking	t	-.936	.124	-2.007*	.209	-.904	-1.990*	.956	-.478	1.060
Per. II	β	.70452	.77656	.67082	.69536	.70768	.46721	.73878	.64437	.71022
Pulmonary	t	30.554 [#]	38.026 [#]	27.759 [#]	29.741 [#]	30.688 [#]	16.188 [#]	33.685 [#]	25.835 [#]	30.952 [#]
Function										
Adjusted R ²		.50000	.60994	.46241	.49207	.50128	.21761	.55405	.41459	.51797

* p < 0.05

** p < 0.02

*** p < 0.01

**** p < 0.005

p < 0.00005

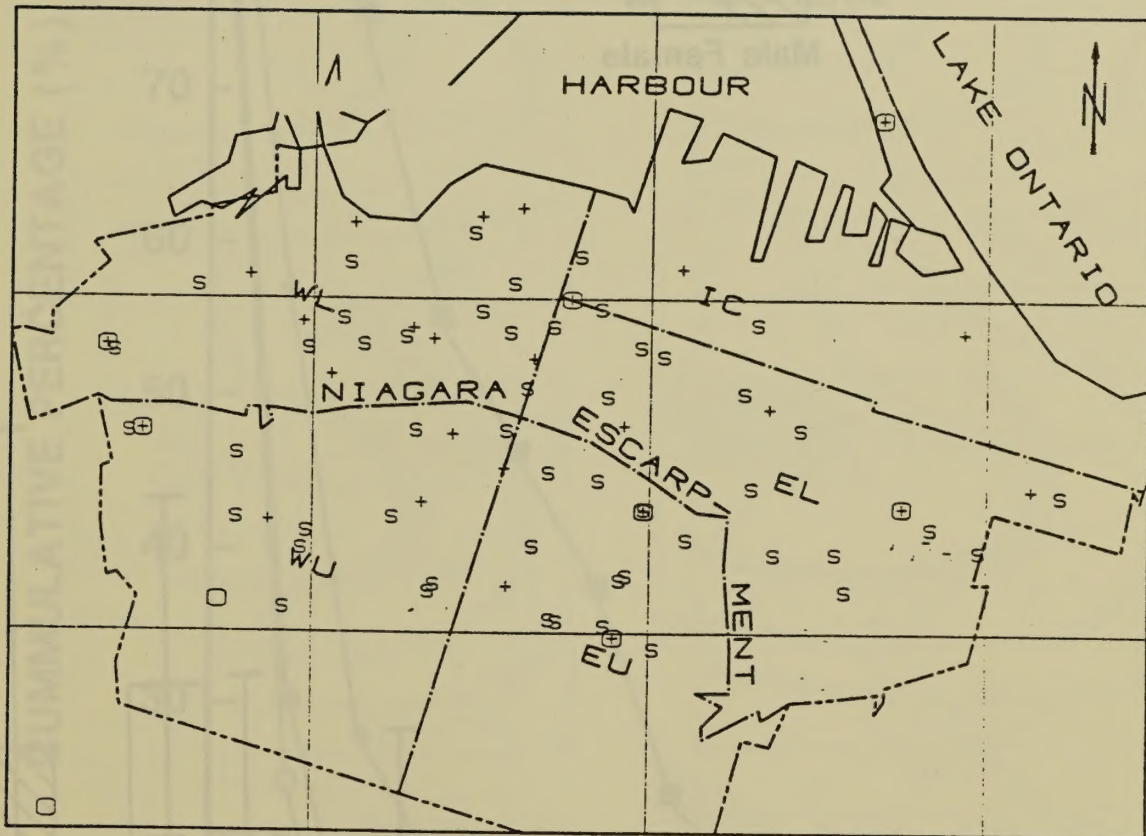
Period IV pulmonary function as dependent variable.

Stepwise deletion of selected Period IV questionnaire and air quality variables.

Period IV self-smoking and Period II pulmonary function as independent variables in each analysis.

For further detail, see p. 58.

FIGURE 2.1



Outline map of Hamilton, Ontario. Symbols are as follows: S; location of study school, +; TSP sampler site; O; PSD sampler site. Double dot-and-dash line; city boundary, dot and dash line; area boundary.

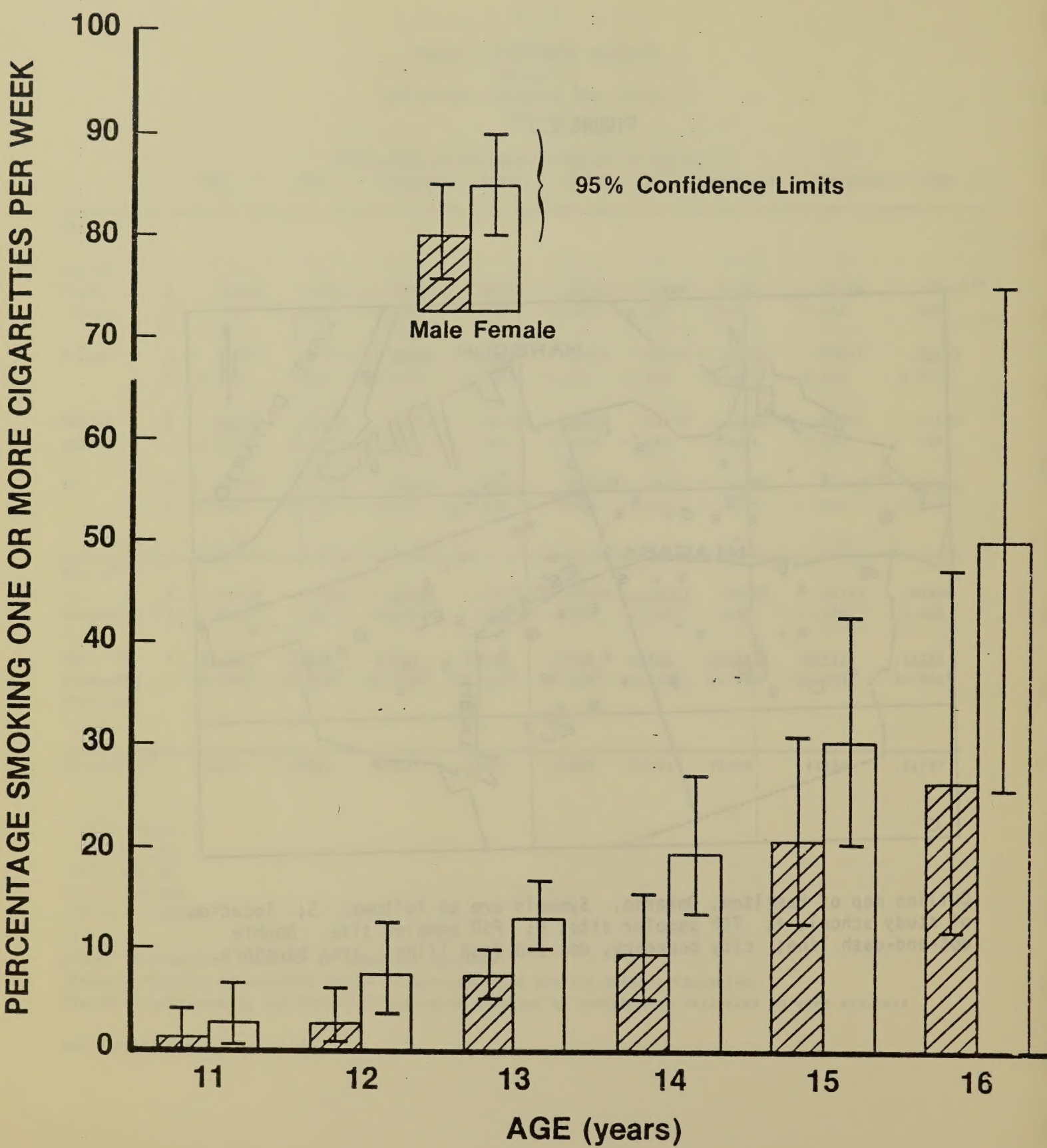


Figure 4.1.1 Prevalence of Smoking as a Function of Age in Period IV

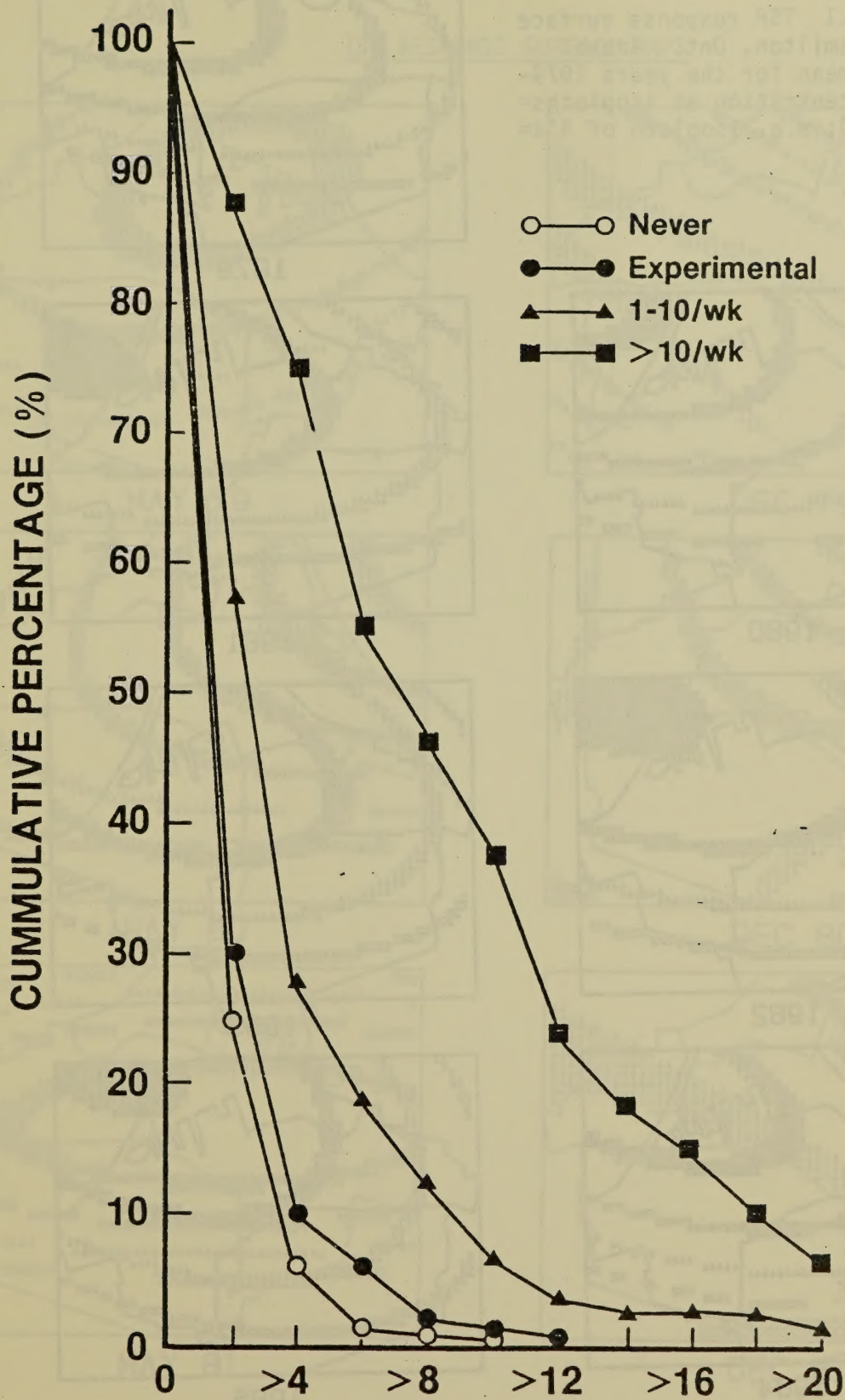
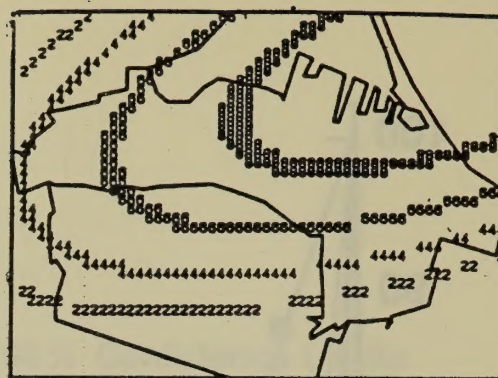
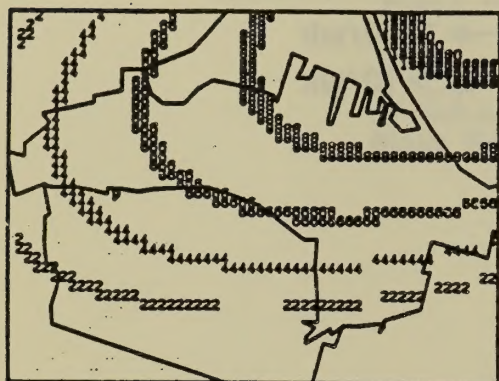


Figure 4.1.2 Difference in CO Concentration Between Expired and Ambient Air (ppm)

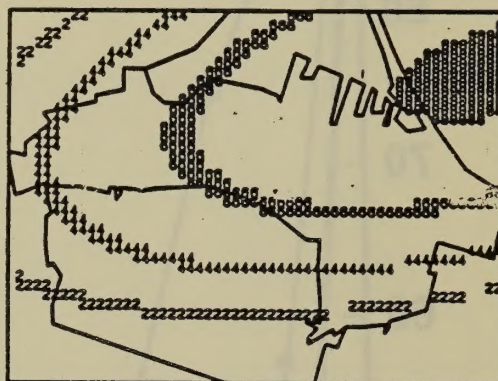
Figure 4.2.1 TSP response surface maps for Hamilton, Ont. Annual geometric mean for the years 1979-1985. Concentration at isopleths = number $\times 10$; e.g. isopleth of 4's = $40\mu\text{g}/\text{m}^3$.



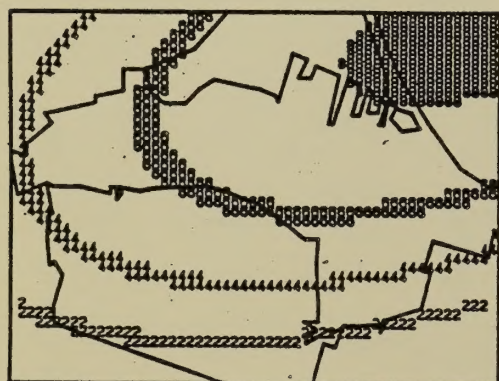
1979



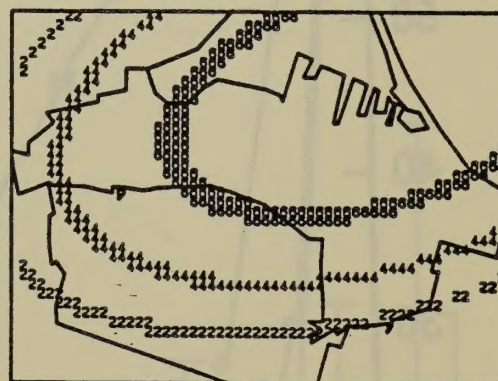
1980



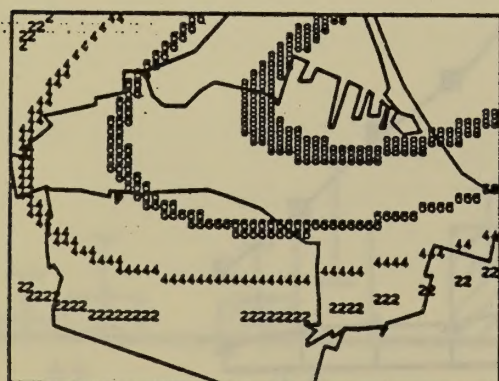
1981



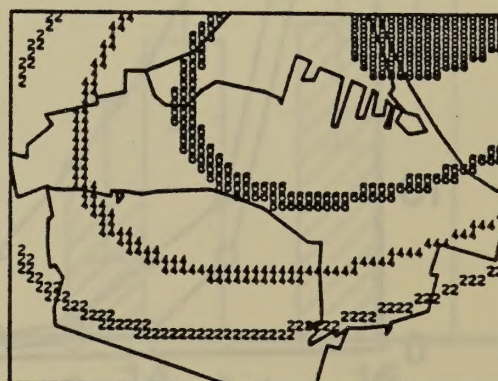
1982



1983



1984

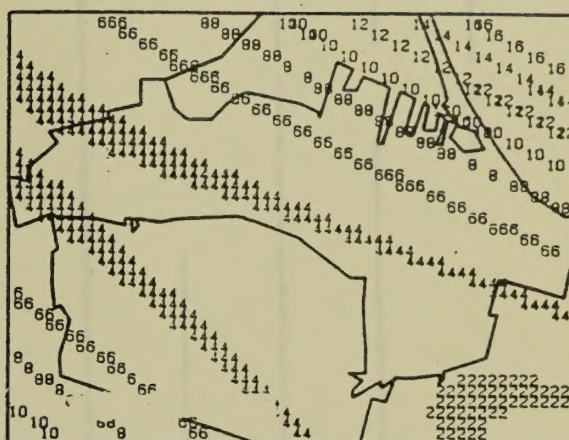


1985

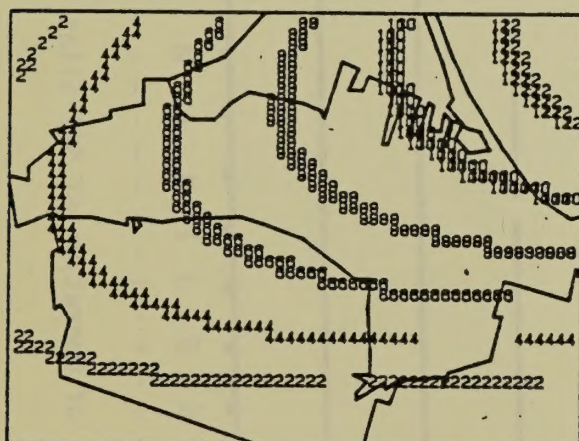
TSP RESPONSE SURFACE MAPS



MAY 79



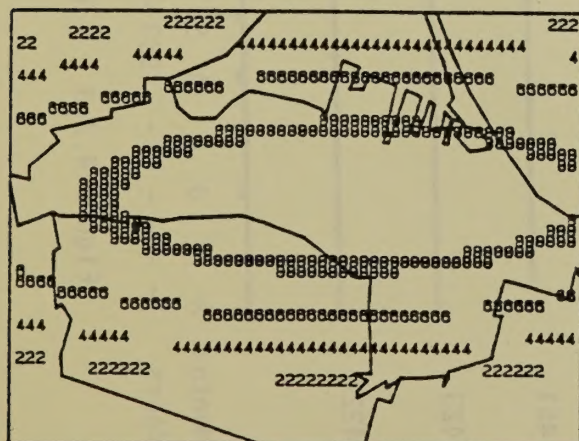
DEC 79



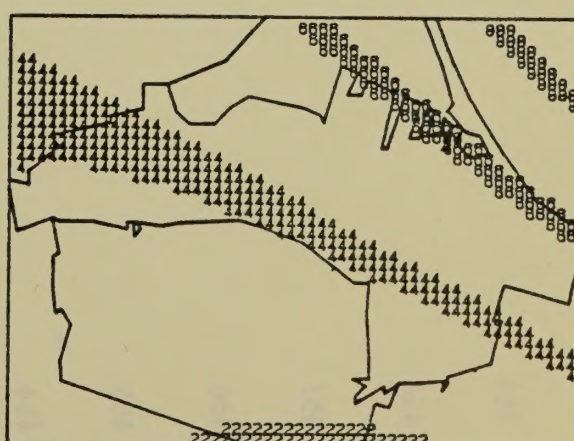
MAY 80



DEC 80



MAY 81



DEC 81

Figure 4.2.2 Concentrations at isopleths = (value shown) $\times 10$

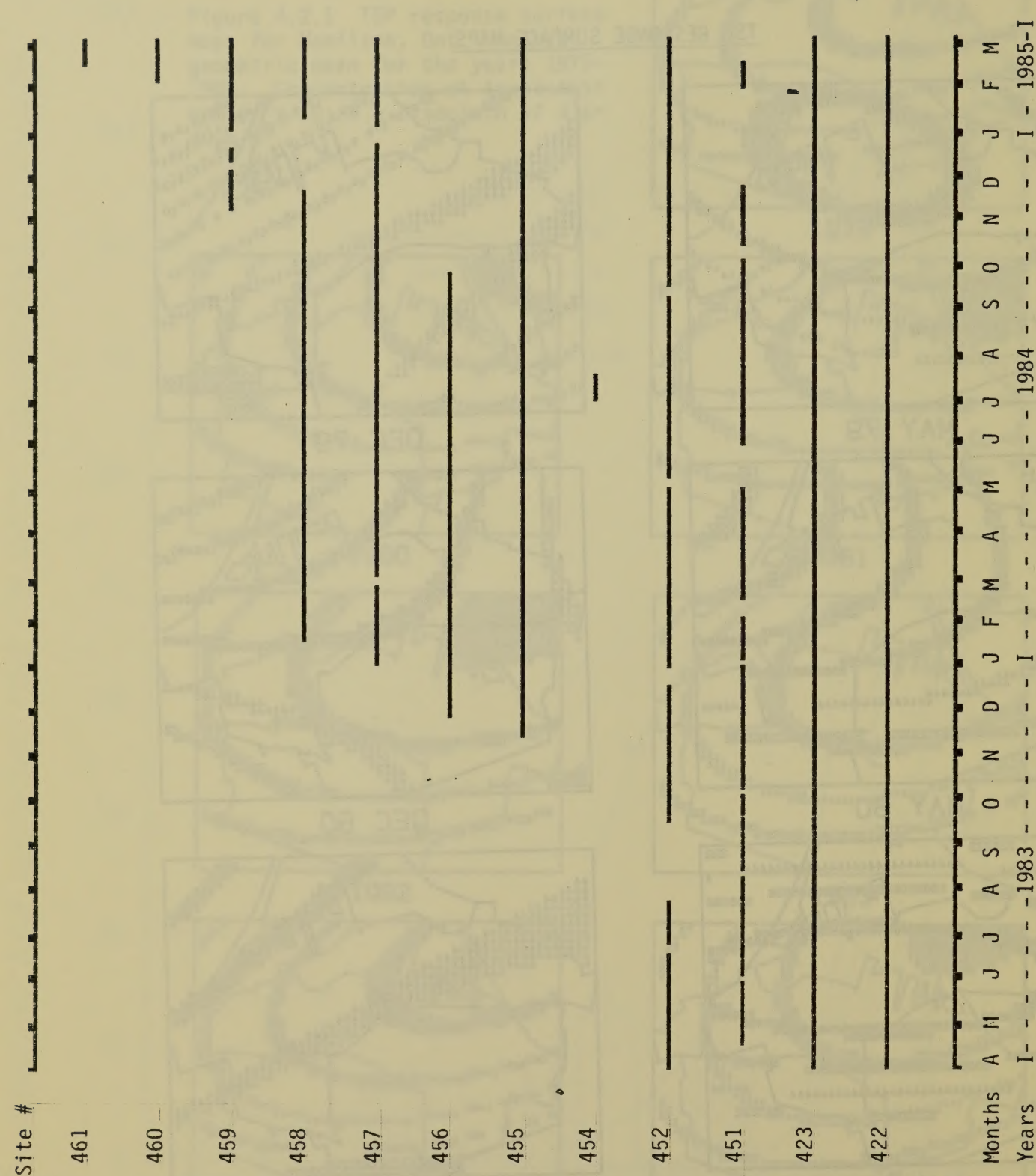


Figure 4.2.3 Times of Operation of SO₂ Sites During Period IV

APPENDIX

FE25-23: High Forced Explosive Flow between 25% and 25% of PVC. Reflects small to medium airway obstruction.

FE25-25: High Forced Explosive Flow between 25% and 25% of PVC. Reflects small to medium airway obstruction.

FE25: See FE25.

APPENDICES:

FE25: Time Forced Explosive Flow. The values of air flow are in a time line, specified by a percentage from the start of expiration, such as FE25 or FE25. Reflects large and large airway obstruction.

GLOSSARY

FE25: Time Forced Explosive Flow. The values of air flow are in a time line, specified by a percentage from the start of expiration, such as FE25 or FE25. Reflects large and large airway obstruction.

LIST OF PUBLICATIONS AND COMMUNICATIONS

FE25: Time Forced Explosive Flow. The values of air flow are in a time line, specified by a percentage from the start of expiration, such as FE25 or FE25. Reflects large and large airway obstruction.

MANUSCRIPT OF REFERENCE 23

FE25: Time Forced Explosive Flow. The values of air flow are in a time line, specified by a percentage from the start of expiration, such as FE25 or FE25. Reflects large and large airway obstruction.

MANUSCRIPT OF REFERENCE 24

FE25:

FE25: See FE25.

FE25: Time Forced Explosive Flow. The values of air flow are in a time line, specified by a percentage from the start of expiration, such as FE25 or FE25. Reflects large and large airway obstruction.

FE25: Time Forced Explosive Flow. The values of air flow are in a time line, specified by a percentage from the start of expiration, such as FE25 or FE25. Reflects large and large airway obstruction.

FE25: Time Forced Explosive Flow. The values of air flow are in a time line, specified by a percentage from the start of expiration, such as FE25 or FE25. Reflects large and large airway obstruction.

Greater than 7.5 microns;
2.5 to 7.5 microns;
2.5 to 2.5 microns;
1.1 to 2.5 microns; and
Less than 1.1 microns.

GLOSSARY

FEF25-75: Mean Forced Expiratory Flow between 25% and 75% points of FVC. Reflects small to medium airways obstruction.

FEF75-85: Mean Forced Expiratory Flow between 75% and 85% points of FVC. Reflects small airways obstruction.

FEV1: See FEVt

FEVt: Timed Forced Expiratory Volume. The volume of air expired in a given time, specified by t (seconds), from the start of expiration, such as FEV1 or FEV3. Reflects lung size and large airways obstruction.

FEV1/FVC: Ratio of the Forced Expired Volume in one second to Forced Vital Capacity. Reflects obstruction in medium to large airways (volume-corrected).

FVC: Forced Vital Capacity. The maximum volume of air expired following a maximal inspiration, and using a maximal effort to expel the air. Reflects lung size.

MEFx: Maximal Expiratory Flow after x% of FVC has been exhaled. For example, MEF50 is the MEF after the 50% point of FVC has been reached. MEF50 reflects small to medium airways obstruction; MEF75 reflects small airways obstruction.

MEF50: See MEFx

MEF75: See MEFx

MET: Maximal Mid-Expiratory Time. Time in seconds required to exhale from 25% to 75% of FVC. Reflects obstruction in medium to large airways (volume-corrected).

PF: Peak Flow. Maximum rate of expiration during FVC. Reflects lung size and degree of obstruction of large airways.

PSD: Particle Size Distribution. Measurements of distribution of suspended particulate matter aerodynamic diameters, as concentrations of particulate matter in 5 size fractions, by inertial impaction onto perforated metal plates covered with replaceable glass fibre collection discs. Obtained using the High-Volume Four-Stage Cascade Impaction Sampler produced by Andersen 2000, Inc. The size fractions are defined by the 50% ECD of the particles impacting on each sampling disc. These fractions are:

- Greater than 7.0 microns;
- 3.3 to 7.0 microns;
- 2.0 to 3.3 microns;
- 1.1 to 2.0 microns; and
- Less than 1.1 microns.

Spirometry: Measurement of lung volume.

TLC: Total Lung Capacity. The amount of air contained in the lungs following a maximal inspiration.

TSP: Variable name used in analysis. Estimate of child's exposure to suspended particulates, measured as TSP, for a 12-month period. See next entry.

TSP: Total Suspended Particulates. Standard method of determination of outdoor, or ambient, levels of suspended particulate matter for North America. The instrument used for this procedure is a standard, commercially available high-volume (hi-vol) instrument, the General Metal Works, Inc. Model GMWL-2000-H High Volume Air Sampling System. Units: $\mu\text{g}/\text{m}^3$.

$\mu\text{g}/\text{m}^3$ or $\mu\text{g}/\text{m}^3$: Micrograms per cubic metre. Levels of particulate pollution are commonly expressed in these units. Levels of gaseous pollutants may be so expressed.

VC: Vital Capacity. The maximum volume of air exhaled voluntarily, after inhaling maximally. Reflects lung size.

PUBLICATIONS AND COMMUNICATIONS

PUBLICATIONS

Pengelly LD, Kerigan AT, Goldsmith CH, Inman E. "The Hamilton Study: Distribution of factors confounding the relationship between air quality and respiratory health". J Air Poll Contr Assn 34: 1039-1043, 1984.

Kerigan AT, Goldsmith CH, Pengelly LD. "A three-year cohort study of the role of environmental factors in the respiratory health of children in Hamilton, Ontario: Epidemiology survey design, methods, and description of cohort". Amer Rev Respir Dis. 133: 987-993, 1986.

Pengelly LD, Goldsmith CH, Kerigan AT, Furlong WF, Toplack SA. "The Hamilton Study: Effect of particle size on the respiratory health of school children". Aerosols. In Press, 1986.

Norman GR, Pengelly LD, Kerigan AT, Goldsmith CH. "Respiratory function of children in homes insulated with urea formaldehyde foam insulation". Can Med Assoc J 134 (10): 1135-1138, 1986.

SUBMITTED FOR PUBLICATION

Pengelly LD, Goldsmith CH, Kerigan AT, Furlong W, Toplack SA. "The Hamilton Study: Estimating exposure to suspended particulates". J Air Poll Contr Assn. Submitted 1985.

Pengelly LD, Furlong WF, Goldsmith CH, Toplack SA, Kerigan AT. "The Hamilton Study: Indoor/outdoor air quality relationships for homes and elementary schools". J Air Poll Contr Assn. Submitted 1986.

Goldsmith CH: "Human Air Pollution Exposure Estimation for Health Effects, considerations for Local Area Networks, Environmetrics 86 Proceedings". Can J Statist. Submitted 1986.

Kerigan AT, Goldsmith CH, and Pengelly LD. Early cigarette smoking in Canadian adolescents: 1. Description of cohort and survey methods. Can Med Assn J. Submitted 1986.

Kerigan AT, Goldsmith CH, and Pengelly LD. Early cigarette smoking in Canadian adolescents: 2. Effect of smoking on respiratory health. Can Med Assn J. Submitted 1986.

MANUSCRIPTS IN PREPARATION

Pengelly LD, Goldsmith CH, Kerigan AT. "The Hamilton Study: The role of environmental factors in the respiratory health of school children". [American Review of Respiratory Disease.]

Pengelly LD, Kerigan AT, Goldsmith, CH. "The Hamilton Study: Use of Postal Codes to establish spatial location of subjects in an epidemiological study". [Canadian Geographer]

Goldsmith CH, Furlong WJ, Pengelly LD, Kerigan AT, Toplack SA. The Hamilton Study: Quality assessment of a particulate sampling network. [J Air Poll Contr Assn]

"SCHOOL CHILDREN'S STUDY" PROJECT REPORTS

These publications are in the form of reports to granting agencies. The first of these publications is our Pilot Study report, submitted to Health and Welfare Canada in 1977:

1. Kerigan AT, Gschwandtner G, Pengelly LD, Ballik EA, Garside BK, and Shewchun J (1977), "Results of a Pilot Study on the Effects of Suspended Particulates and Sulphur Dioxide on the Respiratory Health of School Children". Report to Health and Welfare Canada (Project 6606-1374-40); September 1977.

The following publications were submitted as progress reports to Health and Welfare Canada (Project 6606-1752-53) and to the Ontario Ministry of the Environment, Air Resources Branch (Project 78-020-33):

2. Kerigan AT, Pengelly LD, Goldsmith CH, and Shewchun J (1979a), "The Effect of Environmental Factors on the Respiratory Health of School Children in Hamilton--Current Report #1, March 1, 1979".
3. Kerigan AT, Pengelly LD, Goldsmith CH, and Shewchun J (1979b), "The Effect of Environmental Factors on the Respiratory Health of School Children in Hamilton--Current Report #2, October 1, 1979".
4. Kerigan AT, Pengelly LD, Goldsmith CH, and Garside BK (1980a), "The Effect of Environmental Factors on the Respiratory Health of School Children in Hamilton--Current Report #3, April 1, 1980". 2 Volumes.

The next three publications were submitted to Health and Welfare Canada and the Ontario Ministry of the Environment, and to the Ontario Ministry of Health (Public Health Grant CHS-R34):

5. Kerigan AT, Pengelly LD, Goldsmith CH, and Garside BK (1980b), "The Effect of Environmental Factors on the Respiratory Health of School Children in Hamilton--Current Report #4, October 1, 1980". 2 Volumes.
6. Kerigan AT, Pengelly LD, Goldsmith CH, and Garside BK (1981a), "The Effect of Environmental Factors on the Respiratory Health of School Children in Hamilton--Current Report #5, April 1, 1981".

7. Kerigan AT, Pengelly LD, Goldsmith CH, and Garside BK (1981b), "The Effect of Environmental Factors on the Respiratory Health of School Children in Hamilton--Current Report #6, October 1, 1981". 2 Volumes.

Additionally, the following Master's Thesis was prepared using data from this study:

8. Kerigan AT (1982), "An Index of Respiratory Disease in Children for Environmental Studies". An M.Sc. Thesis in the Design, Measurement and Evaluation Program, Faculty of Health Sciences: submitted to the School of Graduate Studies, McMaster University, December 1982.

The final report of the "School Children's Study" project has been submitted to the three granting agencies mentioned previously. The full report (9., following) was submitted to these agencies and to selected individuals and groups, while a summary report (10., following) was sent to other individuals and groups.

9. Kerigan AT, Pengelly LD, Goldsmith CH, and Garside BK (1983), "The Hamilton Study: The Effect of Environmental Factors on the Respiratory Health of School Children. Final Report. June 30, 1983". 2 Volumes.

10. Kerigan AT, Pengelly LD, Goldsmith CH, and Garside BK (1983), "The Hamilton Study: The Effect of Environmental Factors on the Respiratory Health of School Children. Final Report--Summary Version. June 30, 1983".

"CHRONIC LUNG DISEASE STUDY" PROJECT REPORTS

The following publications were submitted as Progress Reports to the NHRDP of Health and Welfare Canada (Project 6606-2107-53) and to the Ontario Ministry of Health (Public Health Grant CHS-R64, and MOH Research Grant 00672). Additional copies have been sent to the Ontario Ministry of the Environment, for information."

1. Kerigan AT, Pengelly LD, and Goldsmith CH (1983), "Role of Environmental Factors in the Development of Chronic Lung Disease in Smoking and Non-Smoking Adolescents. Progress Report. November 15, 1983."
2. Kerigan AT, Pengelly LD, Goldsmith CH (1984). "Role of Environmental Factors in the Development of Chronic Lung Disease in Smoking & Non-Smoking Adolescents. Progress Report, November 15, 1984.

COMMUNICATIONS

The following is a list of communications which have resulted from the "School Children's Study", the "Chronic Lung Disease Study", and our "Indoor Air Quality Study", to the end of October 1986. They have been organized into two groups: "Invited"; and "Volunteered". The presenter(s) names have been underlined.

INVITED PRESENTATIONS

1. Inman E. "Evaluation of the Acquisition and Validity of the NWS Test". Proc. (Ann Mtg Can Soc Cardiovasc Pulm Technol) Hamilton, ON, September 1980.
2. Pengelly LD, Kerigan AT, Goldsmith CH, Garside BK. "Effects of the Breathing Environment on the Respiratory Health of School Children in Hamilton, Ontario". Proc. (28th Ann Ont Ind Waste Conf; Ont Min Envt); Toronto, ON, June, 1981.
3. Pengelly LD, Kerigan AT, Goldsmith CH, Furlong WF, Spurgeon WR, Toplack SA. "A multipollutant sampler for indoor and outdoor ambient air". Proc. (Int. Symp. on Indoor Air Poll'n, Health and Energy Conserv); Session C2, No. 11. Harvard Univ: JFK School of Gov't; and School of Public Health. Amherst, MA; Oct. 13-16, 1981.
4. Inman E. "A Comparison of the Forced Vital Capacity Manoeuvre with the Multiple-Breath Nitrogen Wash-Out as a Tool for Measuring Pulmonary Function in an Epidemiological Study Involving Children Aged 7 to 14 Years". Proc. (Ann Mtg Can Soc Cardiovasc Pulm Technol); Niagara-on-the-Lake, Ontario, October, 1981.
5. Pengelly LD, Kerigan AT, Goldsmith CH, Garside BK. "The Hamilton Study: Effect of the Breathing Environment on the Respiratory Health of Children". Proc. (Tech. Transfer Conf. #2; Ont. Min. Envt); Toronto ON, November 1981.
6. Furlong W, and Pengelly LD. "Epidemiological Study". Presentation at Technical Seminar (in-house); Ontario Ministry of the Environment, West Central Region; Hamilton, ON, November 1981.
7. Pengelly LD, Kerigan AT, Goldsmith CH, Toplack SA, Furlong W. "The Hamilton Study: Spatial Distribution of SO₂ and TSP in an Urban Area". Proc. (Ann Conf: Air Poll Contr Assn Ont Sect / Poll Contr Assn Ont); Toronto, Ontario, April 1982.

8. Inman E. "A Method of Cataloguing Data Analyses Using the Computer-Managed List System on the H-P 3000". Proc.(Ann Mtg Can Soc Cardiovasc Pulm Technol); Toronto, ON, October 1982.

9. Kerigan AT, Pengelly LD, and Goldsmith CH. "The Hamilton Study: Effects of Environmental Factors on the Respiratory Health of School Children". Presented at Chedoke-McMaster Hospitals, McMaster University Medical Centre Division, Medical Grand Rounds, 1983-10-27.

10. Kerigan AT, Pengelly LD, and Goldsmith CH. "The Hamilton Study: Effects of Environmental Factors on the Respiratory Health of School Children". Presented at Chedoke-McMaster Hospitals, McMaster University Medical Centre Division, Regional Pediatric Grand Rounds, 1983-11-10.

11. Pengelly LD. "Indoor Air Quality". presented at Family Medicine Rounds, Kitchener Memorial Hospital, Kitchener, Ontario, 1983-11-24.

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EARLY CIGARETTE SMOKING IN CANADIAN ADOLESCENTS

1. DESCRIPTION OF COHORT AND SURVEY METHODS

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Running head: Smoking in Canadian adolescents

ABSTRACT

We have estimated the prevalence of smoking in a cohort of young Canadians, aged 10 - 16, by means of self-administered questionnaire and have validated this reported behaviour by measurements of expired carbon monoxide. Smoking behaviour increases with age in this age interval and is consistently higher in females than in males, the level of regular smoking being almost 50% in females at age 16, although the confidence intervals were wide at this particular age. There was also a positive association between self-reported smoking and maternal smoking with the prevalence of regular self-smoking being doubled when the mother also smoked. Expired carbon monoxide concentration consistently increased with the level of reported smoking, helping to validate the reported smoking behaviour.

Measurements of respiratory symptom prevalence and pulmonary function were also made to estimate the effects of self-reported smoking in this age group. The reproducibility of all the questionnaire information and pulmonary function measurements was

acceptable.

ABSTRACT

Smoking

Adolescents

Validity and Reproduceability

This reported behavior by measurement of cigarette carbon monoxide. Smoking behavior increased with age in this age interval and is consistently higher in females than in males, one level of regular smoking being almost 50% in females at age 16, although the confidence intervals were wide at this particular age. There was also a positive association between self-reported smoking and external smoking with the prevalence of regular self-smoking being doubled when the mother also smoked. Exhaled carbon monoxide concentration consistently increased with the level of reported smoking, helping to validate the reported smoking behavior.

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1. DESCRIPTION OF COHORT AND SURVEY METHODS

Introduction

Cigarette smoking continues to be a major cause of premature morbidity and mortality (1). However, the development of disabling ventilatory impairment is a gradual process and occurs only in a minority of smokers (2). Early subclinical changes in pulmonary function can be detected however, in some older adolescent smokers (3, 4, 5). In addition, an increase in prevalence of respiratory symptoms can also be found in early smokers (3, 4, 5, 6). However, although an increase in prevalence can lead to its own morbidity, it is not clear whether it can lead to ventilatory impairment (2).

For this reason, the earliest effects of smoking need to be studied in order to describe the course of these changes, to identify the smokers at particular risk of respiratory impairment and to dissect out other factors such as secondhand smoke and childhood

respiratory disease, that might increase this risk. Descriptions of smoking behaviour in adolescents have not always related this behaviour to any particular effect. This is true of the Canadian surveys performed so far (7,8). In addition, in the studies of the effects of cigarette smoking, direct validation of smoking behaviour either has not been performed (3,4,6,9,10) or has not been reported (5). We wish to report on the early development of smoking behaviour in Canadian adolescents, using a biochemical method of validation of this behaviour, and in the following paper, report on the effects of this behaviour on respiratory health and pulmonary function.

Design

The sample to be described was one originally drawn in 1978 for a study of the effects of environmental factors on the respiratory health and pulmonary function of school children (11). The sampling frame was all public elementary schools in Hamilton, Ontario. The city was divided into four quadrants; within each quadrant schools were randomly selected until in each quadrant, 800 children from grades 2, 3

and 4 had been included. In the second year of the study, all new children at these schools within the same age interval as the initial cohort were also included in the study. Three new schools from the area of the city with the highest industrial pollution were also added in the second year of the study and all children within these same age intervals as the initial cohort were added. Consent for the study was obtained from a parent of each child. The consent rate in each year of the study exceeded 95%. Each child was studied by a questionnaire administered to a parent by an interviewer and by pulmonary function testing in the school yearly for three years. This particular study concluded in January 1982.

We then wished to continue to study this cohort with special reference to the effects of personal cigarette smoking. Consent was once again obtained from the parents for their child for the remainder of this second study. Of the 2,692 children still available for the study in June 1982, parental consent was obtained for 2,137 (79.4%). This portion of the study lasted for two school years from September 1982 to June 1984 and will be reported in this paper.

All testing of the students was performed in the schools. Prevalence of respiratory symptoms was estimated by a self-administered questionnaire. Prevalence of cough was estimated by positive responses to the questions:

- 1) "Do you usually cough in the morning?",
 - 1A) If yes, "Do you cough like this most mornings for as much as three months in a row each year?",
- 2) "Do you usually cough during the day or night?",
 - 2A) If yes, "Do you cough like this most days or nights for three months in a row each year?"

Prevalence of wheezing and asthma were estimated by positive responses to the questions:

- 3) "Does your chest ever sound wheezy or whistling?",
 - 3A) If yes, "Does it usually sound like this most days or nights?",
- 4) "Has a doctor ever said you have asthma?",
 - 4A) If yes, "Have you ever had to use your asthma medication in the last year?"

Prevalence of lower respiratory infections was estimated by positive responses to the questions:

- 5) "When you have a cold do you usually cough?",
- 5A) "Is it a chest cough as opposed to a throat cough?",
- 5B) "Is it a cough that produces phlegm?",
- 6) "Do you have a cold now, and if not how many weeks ago did you have a cold?",
- 7) "When you had this cold, did you also have a chest cough?"

Smoking behaviour was estimated using a confidential questionnaire, by positive responses to the questions:

"Have you ever smoked?",

If so "Have you smoked at least one cigarette a week for a period of six months?",

If so "How many cigarettes do you usually smoke in a week?"

The student was asked at what age the first cigarette

was smoked and if a regular brand was smoked. The questionnaire also requested information on maternal smoking and the number of other smokers in the household.

Validation of reported smoking behaviour was provided by measurement of expired carbon monoxide. The technique that was followed was modified from that by Vogt (12). In summary, the student inhaled, held the breath for twenty seconds, exhaled roughly a quarter to a third of vital capacity and then collected the remainder in a plastic bag from which, after allowing time for equilibration, the carbon monoxide concentration was measured by a carbon monoxide analyser. Ambient carbon monoxide concentration was also recorded at the same time. To maximize the validity of this self-reported smoking behaviour, we employed the "pipeline" technique previously described by Evans (13). The technique, as originally described, was the "bogus pipeline", in which only part of the sample was informed of the ability of the particular biochemical test to validate reported behaviour. Because Evans showed that giving this information increased the reporting rate, we decided

to inform all participants in the study of the nature of the test, and specifically, that it would identify recent smoking.

Pulmonary function testing was performed in the school. The apparatus used both in the present and in the previous study was a Hewlett-Packard pneumotachograph based Pulmonary Calculator System HP47804A. In this system, flow is measured by the pneumotachograph and volume is derived by integration of the flow over time. Computations were performed internally and a graphic output of records were obtained for each child at the time of testing.

At the time of testing, the tester also recorded and coded the presence of upper or lower respiratory infection so as to allow analyses to take the presence of these infections into account.

The reproducibility of both the questionnaire information and the pulmonary function testing was estimated by selecting randomly five per cent of the subjects within each school and retesting these subjects at the conclusion of the period of study in

that school. All tests, including the respiratory symptom and smoking questionnaires, the carbon monoxide test and the pulmonary function test were repeated on that occasion.

Methods

When the tester entered a new school, she would meet with all the students and explain in particular the purpose of the carbon monoxide test. During testing in each school, temperature, barometric pressure and humidity were recorded twice daily to be used in computation of the pulmonary function variables. In addition, both the carbon monoxide analyzer and the pneumotachograph were calibrated twice daily. For each student, the respiratory symptom questionnaire was administered first. The student was then weighed and measured using a standard stadiometer with the student in stocking feet. The tester would then re-emphasize to the student the purpose of the carbon monoxide test. The subject would then be asked to inhale, breath hold for twenty seconds and then exhale into a bag. A CO analyser (Gas-Tech, HEC-231), was

used to measure the concentration of CO in the expired gas sample.

Pulmonary function was then measured, with the student standing and non-supported. The subject performed a forced expired maneuver from total lung capacity to residual volume followed by an equally rapid inspiration to total lung capacity. Vital capacity maneuvers were recorded and, to be acceptable, at least two forced vital capacities had to be within 5% of each other and the FEV1 derived from those tests had to be within 10% of each other. Computations were performed internally and a graphic output of the record was obtained for each child at the time of testing. For the purpose of measurement, the computer selected the test with the largest sum of FVC plus FEV1. Both inspiratory and expiratory flow-volume loops were obtained. From these flow-volume loops, the following variables were also computed; peak flow, MEF50, MEF75, FEF25-75, FEF75-85, MET. (see glossary)

Statistical analysis was performed using sub programs in the Statistical Package for the Social Sciences (14), including descriptive analyses, comparison of

sample means with student's t test and comparison of observed with expected frequencies with chi-square statistics. Calculation of confidence intervals for estimates of prevalence was performed by calculating binomial confidence intervals, given the number of successes and the number of trials, by first relating the binomial distribution to the incomplete beta function (15) and then solving two incomplete beta function equations (16), using computer algorithms (References available from the authors). Adjustment of the level of significance for multiple comparisons was performed by the method of Tukey (17), in which the conventional level of significance (0.05) is divided by the number of questions being investigated. Correction of measures of agreement for agreement due to chance was performed by Cohen's Kappa (18).

Results

The age and gender characteristics of the cohort studied are shown in in Table 1. There is an almost equal age distribution of male and female students. At the time of testing the age of the cohort varied from 10 to 16 years with a mean of 12.9.

Because there had been an attrition of the original cohort since 1979, we compared the prevalence of certain characteristics in 1979 of those that had been lost to follow-up, and of those remaining in the study. This comparison is shown in Table 2. Using the method of Tukey for multiple comparison (17), the significance level for the 9 comparisons shown is at the 0.006 level. There were significant differences between the two groups for the prevalence of maternal coughing and smoking and for the occurrence of a serious chest infection before the age of two. In addition, there were significantly more lower income families (annual income less than \$10,000 per year) in the group lost to follow-up. However, there were no significant differences in the prevalence of respiratory symptoms in the child nor of paternal cough or smoking. In addition, but not shown here, there were no significant differences in baseline pulmonary function characteristics.

The prevalence of smoking in each age category is shown in Table 3. The prevalence of heavy smoking (greater than 10 per week) rises from 0% at the age of

11 to 22.7% at age 16. Similarly, for regular smoking (greater than 1 per week), the prevalence rises from 1.6% at age 11 to 36.4% at the age of 16. Figure 1 shows the differences in prevalence of regular smoking between males and females in each age group. There is an excess of females, who smoke over males in each age group above the age of 12, becoming a difference of 23% at age 16. The 95% confidence limits on these estimates of prevalence are also shown on Figure 1. In the age groups 15 and 16 years, the confidence limits are wide due to the relatively small numbers. However, even taking the lower bound of these intervals into account, a steady rise in the prevalence of smoking with age can still be seen.

Table 4 shows the mean difference in carbon monoxide between expired and ambient air in the four smoking categories. The difference in the mean CO difference is least between the never and experimental smoking groups although this difference is significant ($t=3.54$, $p<0.001$). However, the mean CO difference is greater in the group smoking between 1-10/week and greatest in the group smoking more than 10/week. These differences are significantly greater ($p<0.001$)

than the mean CO difference in the never smoking group. In addition the CO difference in the group smoking more than 10/week is significantly greater ($t=6.46$, $p<0.0001$) than the group smoking between 1 and 10/week. The differences between the groups are shown in Figure 2 which shows the the cumulative distribution of carbon monoxide difference for the smoking categories. The distributions for the never smokers and experimental smokers appear to be identical but the differences between these two groups and the other two smoking categories can clearly be seen on this graph. This figure allows immediate comparison of the four groups for any given cut point of CO difference . For example, if one takes the level of four parts per million (ppm) carbon monoxide, only 6% of the never smokers had a level above this in contrast to 75% of the group reporting more than 10 per week. The use of this information in validation of the reported smoking behaviour will be discussed in the following section.

Table 5 shows the relationship between maternal smoking and personal smoking. The presence of

maternal smoking is associated with a significantly ($P < 0.05$) higher prevalence of regular smoking in both the smoking categories. Combining both these categories, 16.1% of those adolescents, whose mother smoked, were regular smokers in contrast to only 7.0% where the mother did not smoke.

Table 6 shows the reproducibility of the questionnaire data. Using the criteria of Landis (18), any measure of chance corrected agreement (estimated Kappa) between 0.61 and 0.80 is substantial and greater than 0.80 is almost perfect. Using these criteria, therefore, the agreement between initial and repeat responses to the questionnaire is at least substantial. Table 7 shows the details of the repeatability of the pulmonary function variables. In no case was the repeat estimate significantly ($p < 0.05$) different from the initial estimate.

Discussion

The prevalence rates for smoking obtained in the study are consistent with those obtained from other North American studies although there are certain

differences (7, 8, 19). Comparisons between studies is made more difficult by differences in methodology. Brown (8) reported an increase in prevalence of smoking with age, rising most sharply between ages 13 to 16, a finding which we have confirmed. They also reported increased smoking in females, whose prevalence of smoking at age of 15 was 27% in contrast to 19% in males. We have observed a comparable difference between sexes, although in our study, the regular smoker was defined differently: one who smoked more than one per week. However, more than 50% of this latter group smoked more than 10 per week. Morison (7) in the other Canadian study published, has estimated the point prevalence of cigarette smoking of students in grades 5 to 12 in four cross-sectional surveys. The prevalence of smoking, (that is one or more cigarettes per week) in 1980 in grades 7 to 9 was 15% for males and 20% for females, results close to those of Brown (8) and ourselves. In addition, he also documented a declining prevalence of smoking over the course of the four surveys. Whether this decline was real, or merely due to under-reporting is unclear. As in Brown's study, no biochemical validation of smoking data was obtained. Pechacek (19) has

expressed doubt as to how one can interpret a decline in reported rates as indicative of a true decline in smoking without additional biochemical validation of smoking behaviour.

Several studies from the United States have used biochemical methods of validation. Pechacek (19) has used measurements, both of expired carbon monoxide and of salivary thiocyanate to validate the reports of smoking behaviour. In addition, he used, as we did, the pipeline technique in which all the students are informed as to the purpose of the validation method in order to increase the rate of true reporting. The importance of this pipeline technique on the reporting of smoking behaviour is shown both by the comparability of the smoking rates from his study and ours and by the fact that these rates are higher than other studies which use self-report measures of smoking without the influence of this additional validation method. For example, Tager (4) reports a decrease in smoking prevalence from 37.2% in 1974 to 10.2% in 1982 in males age 15 to 19. They however use no biochemical measure of validation and although the rates for females are comparable to ours and to those

of Pechacek (19), without additional validation one cannot determine the accuracy of this estimate.

The other similarities between our study and that of Pechacek (19) were the data on the level of expired carbon monoxide in different categories of smokers. They reported a value of approximately six ppm for smokers of less than 20 per month in the age interval from 11 to 15 years. In smokers of about 20 a week in the same age interval, CO levels vary from six to 10 ppm. These similarities increase the confidence we have in the accuracy of our estimates of smoking prevalence.

Measurement of expired carbon monoxide has been used to validate reported smoking behaviour in a number of studies, both in adults (11, 20, 22) and in school students (19, 21, 22). Theoretically with its half life of 4 to 5 hours, it should be less useful than thiocynate (half life 10 - 14 days) or cotinine (half life 30 - 45 hours) (19), especially in the population we are studying where smoking is much lighter than in adults. In practice, however, in our hands, it has proved to be extremely useful. It should be stressed,

however that we are not as concerned with individual validation, as with the correct classification of the smoking groups. Only 6.7% of those reporting no smoking had an expired carbon monoxide level greater than 4 ppm compared with 74% of those reporting more than 10 cigarettes per week. We therefore agree with Pechacek (19) that the use of the level of expired carbon monoxide is likely to be as useful as the salivary thiocyanate method of validation although it is possible that the measurement of salivary cotinine will be preferred for individual validation in assessing the efficacy of smoking withdrawal programs in high school students.

However, it is clear from the above that in any smoking study some independent method of validation is necessary. Although no current method is a "gold standard" we do not agree with Pettiti (20) who states that self reports are sufficient in themselves and that it is they that validate the biochemical measures rather than vice versa.

In addition to the use of the pipeline technique, other features in our study probably contributed to

obtaining a rate of reporting as close as possible to the actual smoking prevalence. These features, described by McKennell (23), were the self-administration of the questionnaire, obtaining of answers in schools rather than at home and some, emphasis but not over-emphasis on confidentiality. However, our questionnaires were administered individually and not in a group setting, which additional feature McKennell found to increase the reported prevalence. If our reported rates are valid for the sample studied, are they valid for the sampling frame from which they were originally drawn in 1978. The sample studied was not significantly different in gender, respiratory symptom prevalence, nor in pulmonary function from those lost to followup. However, as seen in Table 2, the group lost to followup had a higher prevalence of maternal smoking. Since this characteristic is associated with a higher rate of students smoking (Table 5), then, if anything, our estimates of smoking prevalence are lower than would be obtained if the entire original sample was surveyed.

An integral part of validity is the reproducibility of

the data. We have estimated this by retesting a subsample within a two week period of the original test. The reproducibility of the questionnaire data, including the information on smoking, was quantified using the chance-corrected agreement statistic Kappa (18), and by this, the agreement is "substantial to almost perfect". Other studies have looked at the reproducibility of questionnaire data on smoking. The study by Woolcock (24) mentions that the agreement was very high but does not state whether the agreement was corrected for that due to chance alone. In a second study, a Kappa statistic of 0.78 was obtained when the reproducibility of an ever smoking history was estimated by resurveying a sample of youths (age 8-17) (25).

Conclusions

In the study we have attempted to obtain reliable and valid data on smoking prevalence in order to relate this smoking behaviour to possible changes in respiratory health and pulmonary function. The data we have obtained have indicated, even allowing for the

somewhat smaller samples in the older age interval, that the prevalence of smoking rises substantially between the ages of 13 and 16 and that the prevalence is much higher for females than for males. We have also shown that the measurement of expired carbon monoxide is a simple and inexpensive method which allows one to biochemically validate the reports of smoking behaviour despite the apparent theoretical limitations in a sample whose smoking is somewhat light and probably infrequent. Having established the reliability and probable validity of these data, a separate paper will then examine whether this behaviour has any effect on respiratory health.

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TABLE 1

AGE DISTRIBUTION OF COHORT

AGE	GENDER		TOTALS
	MALE	FEMALE	
10	0	1	1
11	127	136	263
12	205	179	384
13	464	457	921
14	150	148	298
15	85	76	161
16	27	20	47
TOTALS	1058	1017	2075

NOTES: Age is in years.
Missing observations = 15
(Missing subjects are those for whom
consent was given, but testing was
not performed due to unavailability of
subject.)

TABLE 2
DIFFERENCES BETWEEN SUBJECTS STUDIED IN CURRENT STUDY
AND THOSE LOST TO FOLLOW-UP SINCE 1979

CHARACTERISTIC	PREVALENCE (%)		p-VALUE
	CURRENT COHORT	LOST TO FOLLOW-UP	
MALE	50.1	53.4	0.059
SUBJECT COUGH IN MORNING	5.5	7.0	0.006
SUBJECT EVER WHEEZE	24.9	26.5	0.30
MOTHER COUGH IN MORNING	14.3	21.1	<0.0001
MOTHER SMOKE	39.6	49.6	<0.0001
FATHER COUGH	25.6	26.9	0.44
FATHER SMOKE	44.2	46.1	0.26
INCOME < 10K\$	11.2	23.3	<0.0001
SERIOUS CHEST ILLNESS BEFORE AGE 2	5.9	10.3	<0.0001

Data for comparison have been taken from Period II (1979) data set.

TABLE 3
SMOKING STATUS BY AGE

SMOKING CATEGORY					
n (%prevalence)*					
AGE	NEVER	EXPERIMENTAL	1-10/WEEK	>10/WEEK	TOTAL
10	1	0	0	0	1
11	196(76.6)	56(21.9)	4(1.6)	0	256
12	227(63.2)	114(31.8)	17(4.7)	1(0.3)	359
13	480(53.6)	324(36.2)	61(6.8)	30(3.4)	895
14	141(48.6)	107(36.9)	21(7.2)	21(7.2)	290
15	59(37.6)	58(36.9)	17(10.8)	23(14.6)	157
16	12(27.3)	16(36.4)	6(13.6)	10(22.7)	44
TOTALS	1116(55.7)	675(33.7)	126(6.3)	85(4.2)	2002

NOTES: Age is in years.

Missing observations = 88

*:% prevalence is the percentage based on the
row total, rounded to one decimal place.

TABLE 4

DIFFERENCE IN CARBON MONOXIDE LEVEL BY SMOKING CATEGORY

SMOKING CATEGORY	NUMBER	CO DIFFERENCE* (mean, ppm)	STANDARD DEVIATION
NEVER SMOKED	1116	2.22	1.77
EXPERIMENTAL	659	2.55	2.13
1-10 CIG/WK	125	4.35	4.27
>10 CIG/WK	85	9.78	7.87
TOTAL	1985		

*Difference in ppm between carbon monoxide concentrations in expired air and in ambient air.

Missing cases = 105

TABLE 5

RELATIONSHIP BETWEEN MATERNAL AND PERSONAL SMOKING

	MOTHER SMOKING		TOTAL
	YES	NO	
PERSONAL SMOKING			
0*	637(83.8)**	1101(93.0)	1738
1-10/WK	70(9.2)	51(4.3)	121
>10/WK	52(7.0)	32(2.7)	84
TOTAL	759	1184	1943

NOTES: * Includes experimental smoking
 ** Column percentage

Missing observations = 149 147
 $\chi^2 = 40.60$ $p < 0.001$

Entries in core of table are of form:
 n (prevalence %)

"Total" entries are of form: n

TABLE 6
REPRODUCIBILITY OF QUESTIONNAIRE DATA

QUESTION	n	MISSING	RAW AGREEMENT	CHANCE-CORRECTED AGREEMENT (K)
COUGH IN MORNING	146	4	0.95	0.81
COUGH DAY OR NIGHT	146	4	0.90	0.70
CHEST EVER SOUND WHEEZY	146	4	0.90	0.73
PREVIOUS HISTORY OF ASTHMA	146	4	1.0	1.0
COLD USUALLY ACCOMPANIED BY COUGH	146	4	0.93	0.78
GAS COOKING	146	4	0.98	0.93
HISTORY OF EVERY SMOKING	141	9	0.99	0.97
HISTORY OF CURRENT SMOKING	62*	3	0.95	0.87
MOTHER SMOKE	138	12	1.0	1.0
OTHERS IN HOUSE SMOKE				

*This question only answered by those with a positive response to the previous question (ever smoked).

TABLE 7

REPRODUCIBILITY OF PULMONARY FUNCTION DATA

VARIABLE*	n	INITIAL TEST** (MEAN, SD)	REPEAT TEST** (MEAN, SD)	t-VALUE ⁺	p-VALUE
FVC	150	3.36, 0.75	3.38, 0.75	-1.85	0.07
FEV1	150	2.73, 0.61	2.74, 0.60	-0.31	0.76
$\dot{V}_{50}$	150	3.45, 0.98	3.46, 0.96	-0.47	0.64
$\dot{V}_{75}$	150	1.64, 0.63	1.62, 0.64	1.01	0.31
MET	150	0.59, 0.17	0.59, 0.18	-0.87	0.38
PF	150	5.29, 1.26	5.30, 1.11	-0.11	0.91

* See glossary for description of abbreviations

** Values rounded to two decimal places

+ Significance of difference between variable means in smoking and non-smoking groups.

Figure 1: Relationship between prevalence of smoking one or more cigarettes per week and age in both males and females.

Figure 2: Frequency distribution of difference in CO concentration between expired and ambient air by smoking category.

PERCENTAGE SMOKING ONE OR MORE CIGARETTES PER WEEK

100

90

80

70

60

50

40

30

20

10

0

11

12

13

14

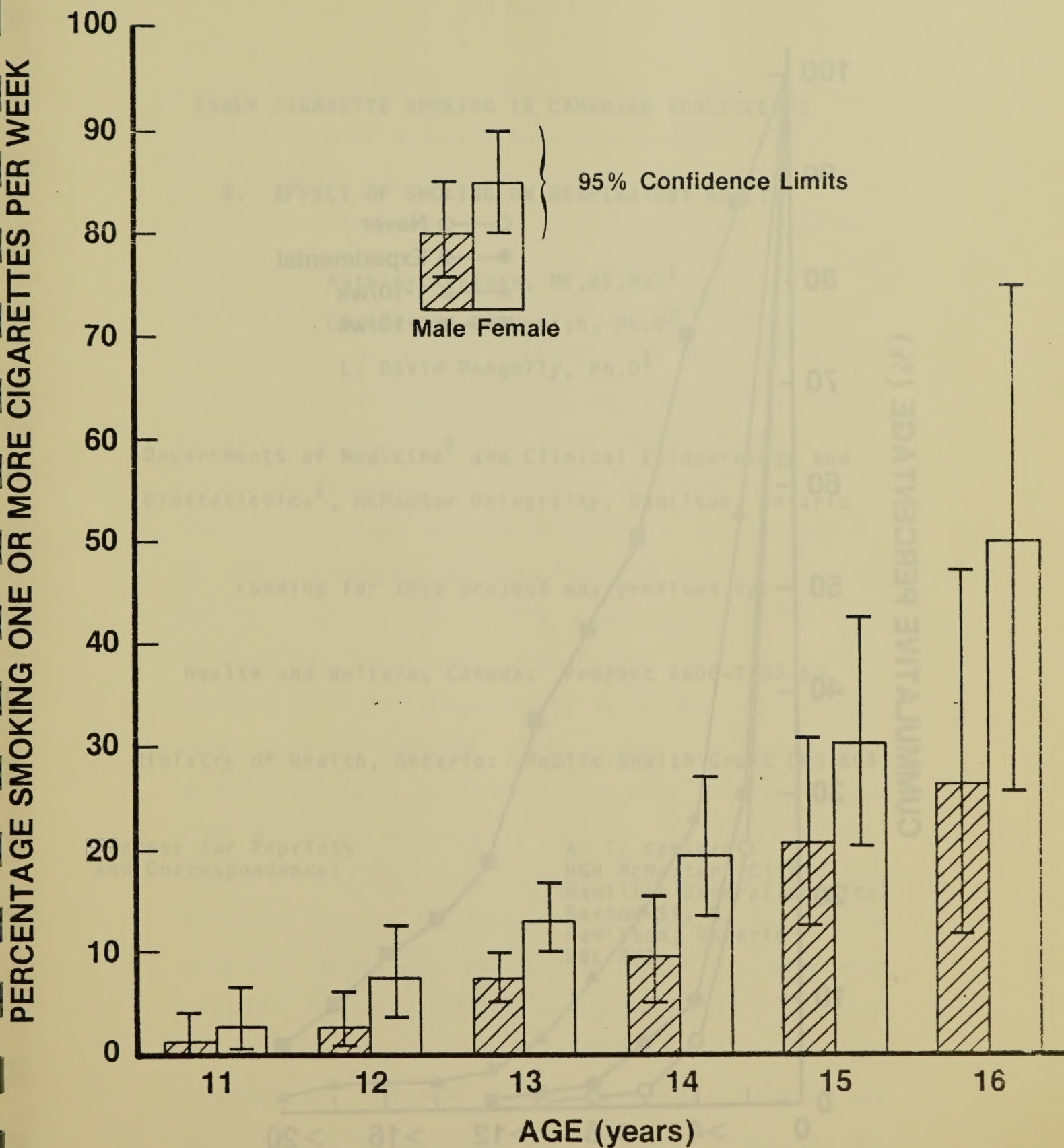
15

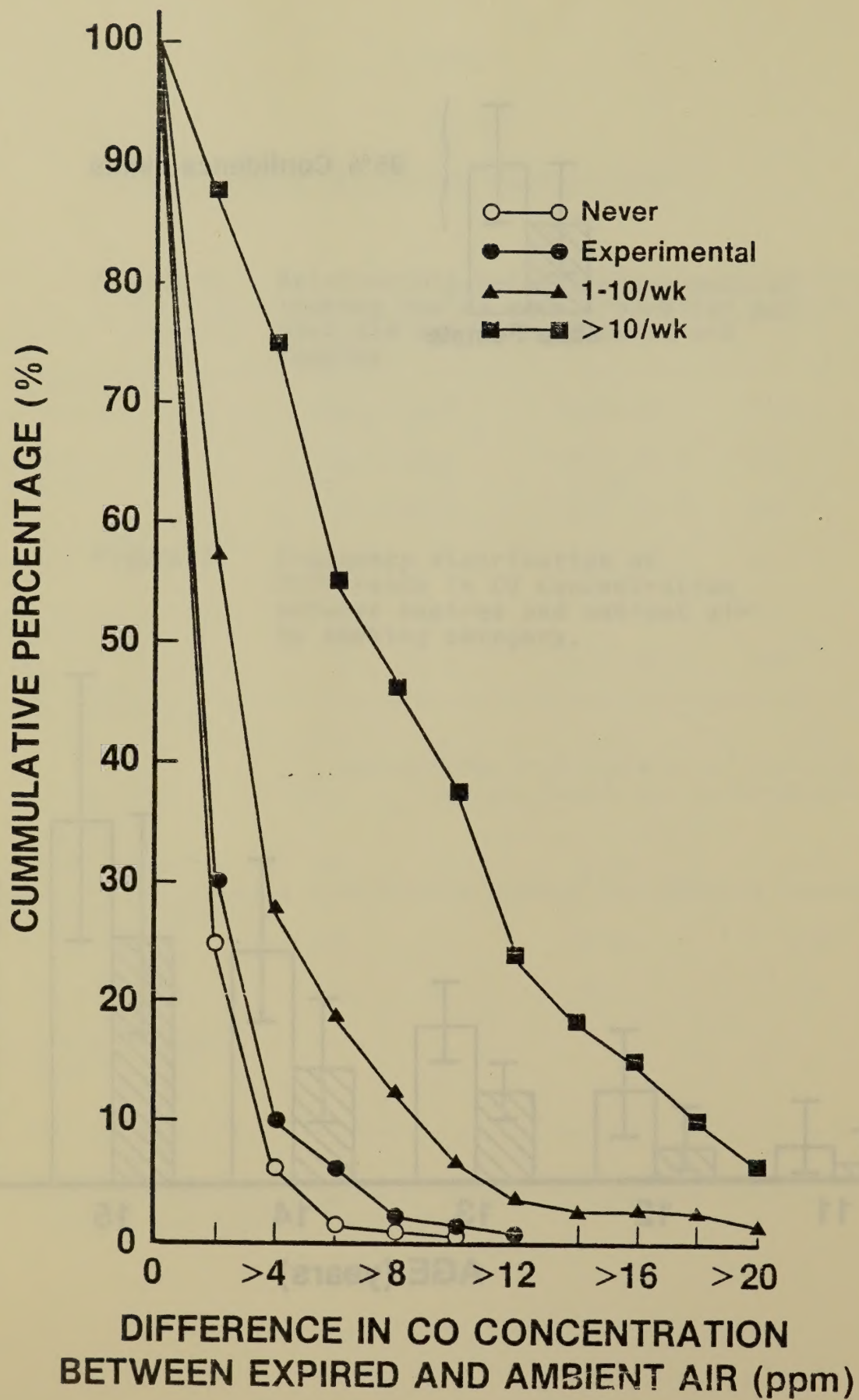
16

AGE (years)

Male Female

95% Confidence Limits





EARLY CIGARETTE SMOKING IN CANADIAN ADOLESCENTS

2. EFFECT OF SMOKING ON RESPIRATORY HEALTH

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ABSTRACT

We have measured the effects of early cigarette smoking in 2090 Canadian adolescents aged 10-16. The prevalence of cough, both in the morning and during the day or night showed the most significant ($p < 0.001$) positive association with the amount smoked in both males and in females. The prevalence of wheezing also increased in a similar fashion. The strength of the relationship between wheezing and smoking was not as great as for cough and only the prevalence of persistent wheezing in females showed a significant relationship with smoking ($P < 0.005$). In contrast to the other symptoms, the prevalence of asthma tended to decrease with increasing smoking although the differences were not significant ($p > 0.05$). The prevalence of a chest cough with the most recent respiratory infection also showed a significant positive association with self-smoking in females and with maternal smoking in males. No other highly significant association between maternal smoking and any other symptom was noted. There was no association however, shown between any decrement in pulmonary function and self-reported smoking and only a possibly significant association ($p < 0.05$) between maternal smoking and FEV1/FVC ratio in males. There was however, a significantly positive association

($p < 0.001$) between FVC and maternal smoking in both males and females, a finding which may be related to the increased lung volume measured in current female smokers, compared to current non-smokers, before the onset of personal smoking.

Smoking

Adolescents

Respiratory symptoms

Pulmonary function

EARLY CIGARETTE SMOKING IN CANADIAN ADOLESCENTS

2. EFFECT OF SMOKING ON RESPIRATORY HEALTH

Introduction

We have described in the previous paper, the patterns of smoking found in the cohort of young Canadian adolescents that had been followed from childhood (1). In this paper, we describe the changes seen, during the earliest years of smoking in pulmonary function and in the prevalence of respiratory symptoms. A number of studies have described the changes seen in smoking adolescents of respiratory symptoms (2-7) and in pulmonary function (2, 5-9). However, in none of these studies was there validation of the reported smoking behaviour. Several of these studies have examined the additional contribution of early childhood respiratory illness (8) and of concurrent parental smoking (9, 10). Although most of these studies have examined cross-sectional differences, two studies have followed subjects prospectively and reported on the longitudinal changes in lung function in smokers and nonsmokers (5,7). In this paper, we

report on the cross-sectional differences in respiratory health between nonsmoking and smoking adolescents and relating these differences to the possible additional effect of maternal smoking.

METHODS

The characteristics of the cohort studied, the measurement of pulmonary function and prevalence of respiratory symptoms and the estimation and validation of smoking behaviour have been described in the previous paper (1). In brief, a cohort of 3600 children, assembled in 1978 have been followed until 1984 when, in conjunction the repeated measurement of pulmonary function and of the prevalence of respiratory symptoms estimates of smoking prevalence were also made by self-administered questionnaire. Validation of the reported smoking behaviour was performed by measurement of expired carbon monoxide. Of the original cohort, 2190 (60.8%) were available for study. For the purpose of this analysis, current smokers have been defined as those smoking one or more cigarettes per week. This group is further sub-divided into light smokers (1-10 per week) and heavy smokers (greater than 10 per week). Experimental smokers, that is those who had smoked but not on a regular basis, were classified with those who had never smoked into the nonsmoking category. Maternal smoking is defined by a positive response to

the question "Does your mother smoke?" in the self-administered smoking questionnaire.

Descriptive analyses, comparison of observed with expected frequencies and examination by multiple regression of the effect of one or several independent variables on a continuously distributed dependent variable were performed by sub-programs in the Statistical Package for the Social Sciences (11). Whenever the dependent variable was discrete, the effect of independent variables was examined by logistic regression analyses using a sub-program in BMDP (12). The overall level of significance was 5%. Adjustment of level of significance for multiple comparisons was performed by the method of Tukey (13). In this case, there were 10 symptoms and 7 pulmonary function variables so the adjustments produced significance levels of 0.005 and 0.007 respectively. All pulmonary function variables, with the exception of FEV1/FVC and MET, (see glossary) were adjusted for height by regression equations developed for males and females separately.

RESULTS

Table 1 shows the prevalence of symptoms by gender. Although in most cases the prevalence is similar, in females there is a significantly ($p < 0.01$) higher prevalence of a day or night cough. In addition, in males the prevalence of a history of asthma is significantly higher ($p = 0.01$).

Table 2 shows the prevalence of these same respiratory symptoms by the level of reported smoking behaviour. The reported prevalence of respiratory symptoms increases with increasing amount of smoking except asthma. The differences between nonsmokers and heavy smokers are most marked for the questions on cough and for that on current wheeze. The relative association of self-reported and maternal smoking and the prevalence of respiratory symptoms is shown in more detail in Table 3 for four of the symptoms which were found to have the most significant association with self-reported smoking and maternal smoking (Table 4). (Data for the other symptoms are available from the authors.) It will be seen that the increasing prevalence of symptoms, with self-smoking is seen both

when the mother is smoking and when she is not but that the effect of maternal smoking overall is much less than that of self-reported smoking.

Table 4 shows the results of logistic regression for males and females separately. These analyses were performed for each gender because of the possibility that there might be differences. Since we were performing multiple comparisons, we adopted a conservative level of significance of 0.005. For both males and females, self-reported smoking had a significant association with a cough during the day or night. In addition, for females only, self-reported smoking had a significant association for current wheezing and also for the occurrence of a chest cough with the most recent respiratory infection. Maternal smoking appeared to have a significant association for this last symptom in males. For no symptom was there found any interaction between self-reported smoking and maternal smoking when an interaction term was used in the logistic regression. Using the less conservative level of significance, that is <0.01 , both maternal and self-reported smoking in females have an association with the prevalence of a morning

cough.

Table 5 shows the results of regression analysis in males using each pulmonary function variable adjusted for height where appropriate as the dependent variable and self-smoking and maternal smoking as the independent variables. Table 6 shows the same results for females. The only significant associations ($p < 0.05$) found for self-smoking were between self-smoking and an increase in the FVC and FEV1 in males. This was not seen in females. No impairment of pulmonary function was seen in association with self-smoking. Likewise, maternal smoking showed a significant association with an increase in FVC and FEV1 in both males and females. It also showed a significant association with a reduction of FEV1/FVC ratio in males alone. Using the more conservative level of significance that is 0.007, only the positive association of maternal smoking with FVC remained significant. Since it was possible that the disproportionate presence of asthmatics in the nonsmokers might be biasing these results, the analyses were reported excluding asthmatics but the results were unchanged.

Discussion

This paper describes an association between self-reported smoking in adolescents and an increased prevalence of certain respiratory symptoms. That this was a causal relationship is suggested by the increasing prevalence with increasing cigarette consumption, the only exception being that of asthma, and the consistency with other studies which have reported on the association of respiratory symptoms and smoking (2,3,4,6,7). The only study which has not shown an association between smoking and symptoms has been that of Tashkin (5). However, in that study, the sample was small (33 smokers; 165 nonsmokers) and it is probable that the analysis lacked the power necessary to demonstrate a significant difference if it indeed existed.

Cough is the most commonly surveyed symptom. However, comparison between studies of the prevalence of cough is made difficult by the varying format of the questionnaires used and the wording of the particular questions. The study by Bland (10) used a wording of

the cough question which was the same as ours. In that study, the prevalences of the day or night cough were similar to those observed by ourselves for both smokers (47%) and nonsmokers (20%). They are also similar for the prevalence of a morning cough in smokers (19%) but were much lower (3%) in the nonsmokers. The paper by Bland (10) is also the only one to report on male and female differences and on the effect of parental smoking. However, the rate of smoking, and hence the number available for analysis, was low in the highest smoking groups (<3% smoking more than 1 a day), reducing the reliability of the reported prevalences in these groups. In our study, only the prevalence of day or night cough was significantly higher in females (Table 1) but both males and females did show a highly significant association of smoking with this symptom (Table 4). Morning cough also showed an association with self-smoking and with maternal smoking in females that may be significant ($0.005 < p < 0.01$) but by using the conservative level of probability for multiple comparisons, one can only be less certain of this association than with day or night cough. Neither self-smoking or maternal smoking have a significant

effect on morning cough in males ($p > 0.01$). This finding in males may be a reflection of the small number of male smokers in the age interval studied since the increase in prevalence of cough in the higher smoking group was similar in both males and females, the prevalence in males increasing with smoking from 12.5% to 28.07% and in females from 11.9% to 26.0%. Bland (10) also reported on the additional effect of parental smoking that was significant ($p < 0.01$) but, as in our study, this effect was much less than that due to self-smoking. We have chosen to measure the effect of maternal smoking only since, in our previous study, its effect was far more pronounced than paternal smoking (unpublished observations). Bland (10) also reports a dose-response relationship between the amount smoked and the prevalence of cough, a finding also reported by Seeley (2) and Adams (6).

The prevalence of wheezing also showed an increase with the amount smoked although the effect of smoking was only significant ($p < 0.005$) in females for persistent wheezing. The importance of wheezing as an indicator of underlying air flow obstruction has been reported on previously (14). However, only one study

(7) has reported on this symptom in relation to smoking. This study (7) found a lower prevalence of persistent wheeze among smokers (5.6%) than we observed.

In contrast to the pattern observed in wheezing, the prevalence of asthma fell with smoking, although the differences in prevalence were not significant ($p > 0.05$) either for males or females. The increased prevalence of the history of asthma in males with the male/female ratio of 1.5 is consistent with that described in the literature (15). We also used several questions to estimate the prevalence of acute lower respiratory tract infections especially in relation to upper respiratory infections. This type of question has not been used by any of the studies quoted previously but has been used in other studies (16,17) in the form of "Do colds tend to go the chest?" A significant association ($p < 0.005$) was found between the prevalence of a chest cough with the most recent respiratory infection and personal smoking in females and maternal smoking in males.

In contrast to the consistent effects observed of

personal smoking and to a lesser extent of maternal smoking on symptoms, no such pattern was observed for pulmonary function. The only decrement in pulmonary function that might be important is the association of maternal smoking with a reduced FEV₁/FVC ratio in males. This lack of significant association is in contrast to a number of recent studies which have described a relationship between smoking in adolescents and impairment of pulmonary function (2,5,6,7,8). However, there is a difference in the ages of the samples studied. The mean age of our sample was 12.9 years and this is younger than the samples in the other studies which are 15 years of age or older. In this period of smoking inception, this difference is probably important since Adams (6) found that the duration of smoking had to be 2 years or more for any significant differences to be found in pulmonary function between smokers and nonsmokers. Tager (7) also found that no differences between smokers and nonsmokers were found unless the adolescent was aged 15 or older. In the study by Tashkin (5), the time interval over which change was found was between the ages of 15 and 20. Therefore it may be that in our study, the sample is of yet too

young an age to be showing changes of pulmonary function with smoking.

The second finding of interest is the association between an increased forced vital capacity and maternal smoking in both males and females and, using a less conservative level of significance, with self-smoking in males. This finding of an increased vital capacity in smokers has been described by other authors (5,6,18). The study by Tashkin (5) also showed a very significantly higher V25 in smokers at age 15 with an increased V50 that was of borderline significance ($p=0.052$). One possible interpretation of these findings is that the initiation of smoking is in part due to a process of self-selection whereby those children with the better lungs become the more frequent smokers. While this appears to be intuitively attractive, it must be pointed out that in none of the other studies was the influence of maternal smoking, which we found to show the stronger association with this paradoxical increase in vital capacity, taken into account. The possibility needs to be explored that it is maternal smoking that in some ways is associated with a compensatory increase

in lung function and that the association with self-smoking that is observed is mainly a result of the association between maternal and self-smoking that we have described in our previous paper (1). Unpublished data from our previous study has shown that maternal smoking has a strongly positive association with vital capacity. In addition, smokers in our study had higher lung volumes and flow rates than nonsmokers when measured four to five years previously before the start of smoking at the mean age of 8.3 years (Table 7). This interrelationship of maternal smoking and constitutional factors in the determination of lung volumes and the starting of cigarette smoking is an area that has been little explored and which must at the present time remain a matter of speculation.

Conclusion

We have been able to demonstrate an association between prevalence of a number of respiratory symptoms and the occurrence of smoking in a group of adolescents varying from 10 to 16 years with a mean age 12.8). This has been the first study in which

this association has been described with concurrent biochemical validation of the reported smoking behaviour. An increase in prevalence of these symptoms with increasing amount smoked has also been found in this study. However, no significant association was found between self-reported smoking and decrement in pulmonary function although the present age of this cohort makes it likely that it is still too early to expect this type of change to be observed. Further followup of this cohort might help identify the point at which these changes do occur. An apparent increase in vital capacity in association with parental smoking was also observed, findings that have been previously been associated with self-reported smoking in other studies.

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TABLE 1

PREVALENCE OF RESPIRATORY SYMPTOMS BY GENDER

SYMPTOM	MALE (n=1047), %	FEMALE (n=1008), %	X , P**	TOTAL (n=2055), %
MORNING COUGH*	141, 13.5	160, 15.9	2.38, >0.1	301, 14.6
MORNING COUGH FOR 3 MONTHS	24, 2.3	37, 3.7	3.42, 0.061	61, 3.0
DAY OR NIGHT COUGH	206, 19.7	248, 24.6	7.25, 0.007	454, 22.1
DAY OR NIGHT COUGH FOR 3 MONTHS	39, 3.7	59, 5.9	5.12, 0.022	98, 4.8
EVER WHEEZE	216, 20.6	203, 20.1	0.15, >0.1	419, 20.4
CURRENT WHEEZE	61, 5.8	56, 5.6	0.07, >0.1	117, 5.7
EVER ASTHMA	106, 10.1	70, 6.9	6.63, 0.010	176, 8.6
CURRENT ASTHMA	63, 6.0	45, 4.5	2.49, >0.1	108, 5.3
CHEST COUGH WITH ANY COLD	417, 39.8	429, 42.6	1.58, >0.1	846, 41.2
CHEST COUGH WITH LAST COLD	316, 30.2	295, 29.3	0.09, >0.1	611, 29.7

* Detail of question given in Kerigan (1).

** Significance of difference in prevalence between genders.

Missing Cases = 35

TABLE 2

PREVALENCE OF RESPIRATORY SYMPTOMS BY
ADOLESCENT SMOKING BEHAVIOUR

SMOKING BEHAVIOUR

SYMPTOM	<u>non-smoker</u> (n=1775), %	<u>1-10/wk.</u> (n=125), %	<u>>10/wk.</u> (n=85), %	<u>TOTAL</u> (n=1985), %
MORNING COUGH*	239, 13.5	26, 20.8	25, 29.4	290, 14.6
MORNING COUGH FOR 3 MONTHS	44, 2.5	4, 3.2	11, 12.9	59, 3.0
DAY OR NIGHT COUGH	357, 20.1	43, 34.4	37, 43.5	437, 22.0
DAY OR NIGHT COUGH FOR 3 MONTHS	74, 4.2	8, 6.4	12, 14.1	94, 4.7
EVER WHEEZE	347, 19.5	24, 19.2	30, 35.3	401, 20.2
CURRENT WHEEZE	87, 4.9	8, 6.4	15, 17.6	110, 5.5
EVER ASTHMA	151, 8.5	9, 7.2	5, 5.9	165, 8.3
CURRENT ASTHMA	94, 5.3	5, 4.0	2, 2.4	101, 5.1
CHEST COUGH WITH ANY COLD	713, 40.2	59, 47.2	46, 54.1	818, 41.2
CHEST COUGH WITH LAST COLD	*511, 28.8	46, 36.8	34, 40.0	*591, 29.8

MISSING CASES - 105 (106 for last question)

* Denominator reduced by 1.

TABLE 3

SYMPTOM PREVALENCE IN SELF-SMOKING CATEGORIESSelf-Smoking (entries are n (total) %)MORNING COUGH

MOTHER SMOKING	0/WK	1-10/WK	>10/WK	TOTAL
YES	98(636) 15.4	15(69) 21.7	16(50) 32.0	129(755) 17.1
NO	134(1100) 12.2	10(51) 19.6	9(32) 28.1	153(1183) 12.9
TOTAL	232(1736) 13.4	25(120) 20.8	25(82) 30.5	282(1938) 14.6

DAY OR NIGHT COUGH

	0/WK	1-10/WK	>10/WK	TOTAL
YES	139(636) 21.9	28(69) 40.6	24(50) 48.0	191(755) 25.3
NO	203(1100) 18.5	14(51) 27.5	13(32) 40.6	230(1183) 19.4
TOTAL	342(1736) 19.7	42(120) 35.0	37(82) 45.1	421(1938) 21.7

CURRENT WHEEZING

	0/WK	1-10/WK	>10/WK	TOTAL
YES	34(636) 5.3	7(69) 10.1	7(50) 14.0	48(755) 6.4
NO	51(1100) 4.6	1(51) 2.0	8(32) 25.0	60(1183) 5.1
TOTAL	85(1736) 4.9	8(120) 6.7	15(82) 18.3	108(1938) 5.6

CHEST COUGH WITH LAST COLD

	0/WK	1-10/WK	>10/WK	TOTAL
YES	204(636) 32.1	24(69) 34.8	24(50) 48.0	252(755) 33.4
NO	295(1099) 26.8	18(51) 35.3	10(32) 31.2	323(1182) 27.3
TOTAL	499(1735) 28.8	42(120) 35.0	34(82) 41.5	575(1937) 29.7

Missing values (incomplete information on symptom or smoking) = 152
(153 for chest cough).

TABLE 4

RELATIONSHIP OF SYMPTOM PREVALENCE TO
MATERNAL AND SELF-SMOKING: LOGISTIC REGRESSION ANALYSIS

GENDER

SYMPTOM	MALE SELF-SMOKE	MALE MOTHER SMOKE	FEMALE SELF-SMOKE	FEMALE MOTHER SMOKE
MORNING COUGH*	0.019*	>0.1	0.008	0.008
MORNING COUGH FOR 3 MONTHS	0.07	>0.1	0.016	0.036
DAY OR NIGHT COUGH	<0.0001	0.06	<0.001	>0.1
DAY OR NIGHT COUGH FOR 3 MONTHS	0.054	>0.1	0.016	>0.1
EVER WHEEZE	0.051	>0.1	>0.1	0.03
CURRENT WHEEZE	0.07	>0.1	0.003	0.55
EVER ASTHMA	0.1	>0.1	>0.1	0.07
CURRENT ASTHMA				
CHEST COUGH WITH ANY COLD	>0.1	0.036	0.012	>0.1
CHEST COUGH WITH LAST COLD	>0.1	0.0003	0.002	>0.1

* Probability that improvement in prediction of outcome (symptom prevalence) by entering of term (self-smoke or mother smoke) is due to chance, i.e., p value.

TABLE 5

RELATIONSHIP OF PULMONARY FUNCTION TO SMOKING - MALES

PULMONARY FUNCTION MEASURE	<u>SELF-SMOKING</u>		<u>MATERNAL SMOKING**</u>	
	t	p	t	p
FVC*	2.11**	0.035	3.49	0.0005
FEV1	2.45	0.014	2.01	0.045
FEV1/FVC	0.607	>0.1	-2.06	0.04
$\dot{V}_{50}$	1.85	0.06	0.50	>0.1
$\dot{V}_{75}$	1.86	0.06	-0.07	>0.1
MET	1.04	>0.1	0.10	>0.1
PF	-0.89	>0.10	0.18	>0.1

* FVC: forced vital capacity; FEV1: forced expired volume in 1 second; $\dot{V}_{50}$: flow at 50% vital capacity; $\dot{V}_{75}$: flow at 75% vital capacity; MET: mid-expired time; PF: peak flow

** t-value associated with inclusion of independent variable in regression analysis, with pulmonary function measure as dependent variable. Positive value of t indicates that pulmonary function measure increases in the presence of maternal or self-smoking and decreases with a negative t.

TABLE 6

RELATIONSHIP OF PULMONARY FUNCTION TO SMOKING - FEMALES

	<u>SELF-SMOKING</u>		<u>MATERNAL SMOKING</u>	
	t**	p	t	p
FVC*	2.23	>0.10	3.28	0.001
FEV1	1.32	>0.10	2.10	0.04
FEV1/FVC	-1.72	0.08	-1.45	>0.10
$\dot{V}_{50}$	1.08	>0.10	-0.01	>0.10
$\dot{V}_{75}$	0.37	>0.10	0.03	>0.10
MET	0.40	>0.69	-0.26	>0.10
PF	-1.19	>0.10	0.11	>0.10

* See Table 5 for definitions

** t-value associated with inclusion of independent variable in regression analysis, with pulmonary function measure as dependent variable. See comments on Table 5.

TABLE 7

COMPARISON OF PULMONARY FUNCTIONS OF
SMOKERS AND NON-SMOKERS IN 1980-81

(Entries are Mean, SD)

	NON-SMOKER	MALES SMOKER	p-VALUE	NON-SMOKER	FEMALES SMOKER	p-VALUE
FEV1	1.91, 0.22	1.94, 0.24	>0.1	1.81, 0.21	1.88, 0.25	0.002
FVC	2.41, 0.24	2.46, 0.25	0.08	2.20, 0.24	2.27, 0.31	0.006
$\dot{V}_{50}$	2.37, 0.58	2.41, 0.66	>0.1	2.44, 0.56	2.55, 0.63	0.03
$\dot{V}_{75}$	1.02, 0.33	1.02, 0.33	>0.1	1.04, 0.32	1.10, 0.36	>0.1
n	910	77		837	130	

* Significance of difference between variable means in smoking and non-smoking groups.

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